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                    STN Columbus

FILE 'HOME' ENTERED AT 15:29:58 ON 09 JAN 97

=> file medline hcaplus
COST IN U.S. DOLLARS                 SINCE FILE     TOTAL
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FULL ESTIMATED COST                   0.15     0.15

FILE 'MEDLINE' ENTERED AT 15:30:10 ON 09 JAN 97

FILE 'HCAPLUS' ENTERED AT 15:30:10 ON 09 JAN 97
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=> s regulat? (2a) complement (2a) activat?
L1       124 FILE MEDLINE
L2       147 FILE HCAPLUS

TOTAL FOR ALL FILES
L3       271 REGULAT? (2A) COMPLEMENT (2A) ACTIVAT?

=> s chimer? or hybrid?
L4    129935 FILE MEDLINE
L5    149124 FILE HCAPLUS

TOTAL FOR ALL FILES
L6    279059 CHIMER? OR HYBRID?

=> s l3 and l6
L7        15 FILE MEDLINE
L8        17 FILE HCAPLUS

TOTAL FOR ALL FILES
L9        32 L3 AND L6

=> s l9 not py>1991
<------User Break------>

SEARCH ENDED BY USER

=> s l9 and py>1991
L10       11 FILE MEDLINE
L11       10 FILE HCAPLUS

TOTAL FOR ALL FILES
L12       21 L9 AND PY>1991

=> s l9 not l12
L13        4 FILE MEDLINE
L14        7 FILE HCAPLUS

TOTAL FOR ALL FILES
L15       11 L9 NOT L12

=> dup rem l15
PROCESSING COMPLETED FOR L15
L16        9 DUP REM L15 (2 DUPLICATES REMOVED)

=> d bib ab 1-

L16 ANSWER 1 OF 9  MEDLINE                    DUPLICATE 1
AN  92086832    MEDLINE
TI  Presence of human chromosome 1 with expression of human
    decay-accelerating factor (DAF) prevents lysis of mouse/human
    ***hybrid*** cells by human complement.
AU  Wang M W; Wright L J; Sims M J; White D J
CS  Department of Surgery, University of Cambridge Clinical School,
    Addenbrooke's Hospital, UK..
SO  SCANDINAVIAN JOURNAL OF IMMUNOLOGY, (1991 Dec) 34 (6) 771-8.
    Journal code: UCW. ISSN: 0300-9475.
CY  ENGLAND: United Kingdom
DT  Journal; Article; (JOURNAL ARTICLE)
LA  English
FS  Priority Journals; Cancer Journals
EM  9203
AB  Xenogeneic organs transplanted to phylogenetically distant species
    are subject to rapid destruction mediated by complement. In humans,
    the ***complement***  ***activation***  is  ***regulated***
    by several proteins encoded by a series of closely linked genes (RCA
    locus) located on chromosome 1. The mouse/human  ***hybrid***
    cell line B10 was found to have retained human chromosome 1. FACS
    analysis confirmed that RCA products such as decay-accelerating
    factor (DAF) were expressed on the membrane surface of B10 cells.
    When exposed to human or rabbit complement in the presence of
    'naturally occurring' human anti-mouse antibodies these cells were
    not lysed by human complement but were killed by rabbit complement.
    This effect could be abrogated by addition of anti-DAF monoclonal
    antibody (IC6). The results offer potential for genetic manipulation
    of the human complement regulatory products in animals to overcome
    xenograft hyperacute rejection.

L16 ANSWER 2 OF 9  HCAPLUS  COPYRIGHT 1997 ACS
AN  1992:104049 HCAPLUS
DN  116:104049
TI  Human complement regulatory proteins expressed on mouse A9 cells
    containing a human chromosome 1
AU  Seya, T.; Okada, M.; Hara, T.; Matsumoto, M.; Miyagawa, S.;
    Oshimura, M.
CS  Dep. Immunol., Cent. Adult Dis., Osaka, 537, Japan
SO  Immunology (1991), 74(4), 719-24
    CODEN: IMMUAM; ISSN: 0019-2805
DT  Journal
LA  English
AB  The structural genes of human complement regulatory proteins are
    clustered on chromosome 1 at position q3.2. Human chromosome 1 was
    transferred into a mouse fibroblast cell line, A9 [designated as
    A9(neo-1)], and the surface expression of its gene products
    participating in complement regulation, namely C3b/C4b receptor
    (CR1, CD35), decay-accelerating factor (DAF, CD55), membrane
    cofactor protein (MCP, CD46) and C3d/EB virus receptor (CR2, CD21),
    were assessed using resp. monoclonal antibodies by flow cytometry.
    CR1 became pos. within 7 days of culture. MCP appeared in a small
    population of cells by Day 3 and, together with DAF, began to
    increase on Day 7. CR2 appeared on Day 14. The order of the
    expression was CR1 > DAF = MCP > CR2. On Day 42, however, all
    became neg. except for MCP, which was markedly diminished. These
    human regulatory proteins were specifically assocd. with the
    presence of human chromosome 1, since none of them were expressed on
    human chromosome 12-transferred A9 cells [A9(neo-12)]. Intact A9
    and A9(neo-12) cells activated human complement via the alternative
    pathway. The activation of this pathway was suppressed in the
    A9(neo-1) cells that expressed CR1, DAF and MCP. Slight protective
    activity was still obsd. in the 42-day cultured A9(neo-1) cells
    expressing only trace MCP. These results suggest that human
    complement regulators, expressed via the transferred human
    chromosome 1, can protect heterologous cells from complement,
    overcoming their ability to activate the human alternative pathway.

L16 ANSWER 3 OF 9  HCAPLUS  COPYRIGHT 1997 ACS
AN  1991:447479 HCAPLUS
DN  115:47479
TI  Synthetic peptide inhibitors of complement serine proteases. III.
    Significant increase in inhibitor potency provides further support
    for the functional equivalence hypothesis
AU  Schasteen, Charles S.; Levine, R. Paul; McLafferty, Sharon A.; Finn,
    Rory F.; Bullock, Lea D.; Mayden, Janet C.; Glover, George I.
CS  Biol. Sci. Dep., Monsanto Co., St. Louis, MO, 63198, USA
SO  Mol. Immunol. (1991), 28(1-2), 17-26
    CODEN: MOIMD5; ISSN: 0161-5890
DT  Journal
LA  English
AB  Synthetic peptides based on functionally equiv. (as defined by
    similar patterns of chem. equiv. amino acids) serine protease
    inhibitor (serpin) C-terminal sequences inhibit both classical and
    alternative pathways of complement activation. Inhibition was also
    found with  ***hybrid***  peptides consisting of the cleavage site
    of one serpin (antithrombin III, .alpha.-1-antitrypsin, or
    antichymotrypsin) attached to the short and long functionally equiv.
    protease binding cores of the other two serpins. A  ***hybrid***
    peptide composed of the sequence at the site of cleavage of C4 by
    C1s attached to the long binding core of antithrombin III was
    selective in inhibiting the classical pathway with no effect on the
    alternative pathway at a concn of 10 .mu.M. Extension of the
    functional equivalence hypothesis has produced inhibitors of
    complement activation named genetic and generic +, whose sequences
    differ by 77% or 87%, resp., from those of all three serpin
    sequences. A  ***hybrid***  peptide composed of the antithrombin
    III cleavage site attached to the generic peptide is an inhibitor of
    complement activation at 500 nM, the most potent inhibitor found in
    this study.

L16 ANSWER 4 OF 9  MEDLINE                    DUPLICATE 2
AN  91048265    MEDLINE
TI  Genes for C4b-binding protein alpha- and beta-chains (C4BPA and
    C4BPB) are located on chromosome 1, band 1q32, in humans and on
    chromosome 13 in rats.
AU  Andersson A; Dahlback B; Hanson C; Hillarp A; Levan G; Szpirer J;
    Szpirer C
CS  Department of Genetics, University of Gothenburg, Sweden..
SO  SOMATIC CELL AND MOLECULAR GENETICS, (1990 Sep) 16 (5)
493-500.
    Journal code: UY2. ISSN: 0740-7750.
CY  United States
DT  Journal; Article; (JOURNAL ARTICLE)
LA  English
FS  Priority Journals; Cancer Journals
EM  9102
AB  C4b-binding protein is involved in the regulation of the complement
    system. It is a multimeric protein composed of seven identical
    alpha-chains and a single copy of a unique beta-chain. The latter
    was identified only recently and its structure determined by cDNA
    cloning. Both subunits in C4b-binding protein belong to the same
    superfamily of proteins composed predominantly of tandemly arranged
    short consensus repeats (SCR) approximately 60 amino acid residues
    in length. The gene for the human alpha-chain is known to be located
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in a gene cluster on chromosome 1, band 1q32, which is called the ***regulators*** of ***complement*** ***activation*** (RCA) gene cluster. We have used cDNA probes for both alpha- and beta-chains of human C4b-binding protein to localize their genes with an in situ ***hybridization*** technique. We find the genes for both chains to be located on chromosome 1, band 1q32, in the human. This suggests that the beta-chain gene is also a member of the RCA gene cluster and that the alpha- and beta-chain genes are located close to each other. The cDNA probes for the alpha- and beta-chains also were used to screen mouse-rat somatic cell ***hybrids*** using Southern blotting to localize their genes in the rat. Both the alpha- and beta-chain genes were shown to be located on chromosome 13 in the rat. These are the second and third genes to be located on rat chromosome 13, and the results suggest that the genes for the alpha- and beta-chains together with the gene for coagulation factor V represent a conserved chromosomal region in rat and man.

L16 ANSWER 5 OF 9 HCAPLUS COPYRIGHT 1997 ACS
AN 1989:513649 HCAPLUS
DN 111:113649
TI A scorpionlike structure of IgG plus antigen and complement, its mechanisms, and its important therapeutic consequences
IN Zagyansky, Yuly
PA Fr.
SO PCT Int. Appl., 23 pp.
CODEN: PIXXD2
PI WO 8806837 A2 880922
DS W: AT, AU, BB, BG, BR, CH, DE, DK, FI, GB, HU, JP, KP, KR, LK, LU, MC, MG, MW, NL, NO, RO, SD, SE
RW: AT, BE, BJ, CF, CG, CH, CM, DE, FR, GA, GB, IT, LU, ML, MR, NL, SE, SN, TD, TG
AI WO 88-EP331 880419
DT Patent
LA English
AB A scorpionlike structure is proposed for IgG in its interactions with antigens, complement, and a small (.apprx.2 kilodalton) regulatory protein based on various knowledge including: the direct interaction of Fab fragment with Fc fragment necessary in the case of the signal switch during complement activation; folding of the hinge region in 2; necessity of appearance of genetic evolutionary mechanisms, with a new gene coding for the IgG hinge region in mammals, which permit better regulation of the antibody-antigen reaction; structural changes affecting energy value changes; the understanding that a 2-kilodalton protein plays a ***regulatory*** role in ***complement*** ***activation*** , etc. The structure of IgG with bound small regulatory protein changes upon binding antigen, the small protein detaches form the Fc region upon pptn. of IgG with antigen, and complement C 1q strongly interacts with the new flexible Fc tail. The practical consequences of this structural model include: treating illnesses due to complement fixation and deficiency syndrome by injection of the regulatory protein; adding to a ***hybridoma*** culture medium the regulatory protein of the same species as the monoclonal antibody produced to bind the antibody instead of the regulatory protein of the fetal calf serum in the medium; creation and injection in patients of antibodies prepd. with special hinge regions; injection of membranous Igs with or without antigen fragments and with or without lymphokines for more efficacious activation of B-lymphocytes; treating or preventing AIDS or cancer; etc.

L16 ANSWER 6 OF 9 HCAPLUS COPYRIGHT 1997 ACS
AN 1989:513648 HCAPLUS
DN 111:113648
TI A scorpionlike structure of IgG plus antigen and complement, mechanisms involved, and important medical consequences of this structure
IN Zagyansky, Yuly
PA Fr.
SO Fr. Demande, 17 pp.
CODEN: FRXXBL
PI FR 2609398 A1 880715
AI FR 87-5658 870421
DT Patent
LA French
AB A scorpionlike structure has been established for IgG in its interactions with antigens and complement based on various new knowledge including: the direct interaction of Fab fragment with Fc fragment; folding of the hinge region in 2; necessity of appearance of genetic evolutionary mechanisms, with a new gene coding for the IgG hinge region in mammals, which permit better regulation of the antibody-antigen reaction; structural changes affecting energy value changes; the understanding that a small 2-kilodalton attached protein plays a ***regulatory*** role in ***complement*** ***activation*** ; etc. The IgG structure changes upon binding antigen, the regulatory protein detaches from the Fc region upon pptn. of IgG with antigen, and complement C 1q strongly interacts with the now flexible Fc tail. The practical consequences of this structural model include: treating illnesses due to complement fixation and deficiency syndrome by injection of the regulatory protein; adding to a ***hybridoma*** culture medium the regulatory protein of the same species as the monoclonal antibody produced to bind the antibody instead of regulatory protein of the fetal calf serum in the medium; creation and injection in patients of antibodies prepd. with special hinge regions; injection of membranous Igs with or without antigen fragments and with or without lymphokines for more efficacious activation of B-lymphocytes; etc.

L16 ANSWER 7 OF 9 MEDLINE
AN 88154754 MEDLINE
TI A physical map of the human ***regulator*** of ***complement*** ***activation*** gene cluster linking the complement genes CR1, CR2, DAF, and C4BP.
AU Rey-Campos J; Rubinstein P; Rodriguez de Cordoba S
CS Department of Immunogenetics, New York Blood Center 10021..
NC DK-19631-11
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1988 Feb 1) 167 (2) 664-9.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 8806
AB We report the organization of the human genes encoding the complement components C4-binding protein (C4BP), C3b/C4b receptor (CR1), decay accelerating factor (DAF), and C3dg receptor (CR2) within the ***regulator*** of ***complement*** ***activation*** (RCA) gene cluster. Using pulsed field gel electrophoresis analysis these genes have been physically linked and aligned as CR1-CR2-DAF-C4BP in an 800-kb DNA segment. The very tight linkage between the CR1 and the C4BP loci, contrasted with the relative long DNA distance between these genes, suggests the existence of mechanisms interfering with recombination within the RCA gene cluster.

L16 ANSWER 8 OF 9 MEDLINE
AN 87252916 MEDLINE
TI Decay-accelerating factor. Genetic polymorphism and linkage to the RCA (***regulator*** of ***complement*** ***activation***) gene cluster in humans.
AU Rey-Campos J; Rubinstein P; Rodriguez de Cordoba S
NC DK 19631-11
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1987 Jul 1) 166 (1) 246-52.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 8710
AB We have investigated the genetic relationships between the human decay-accelerating factor (DAF) and a group of complement components including the C3b/C4b receptor (CR1), C4-binding protein (C4bp), and factor H (H), to which DAF is structurally and functionally related. CR1, C4bp, and H were previously demonstrated to be encoded by a cluster of closely linked genes, which we have designated ***regulator*** of ***complement*** ***activation*** (RCA). Southern blot analysis of genomic DNA using a DAF cDNA probe unraveled the existence of restriction fragment length polymorphism (RFLP) for both Bam HI and Hind III restriction endonucleases. Segregation analysis of these polymorphic fragments in families informative for the segregation of alleles at the CR1, C4BP, and H loci (RCA-haplotypes), demonstrated that, in humans, the gene encoding DAF is located within the RCA gene cluster. No recombinants between DAF and C4BP/CR1 were encountered in 32 informative meioses. In addition, in two individuals showing recombination between the CR1/C4BP and H loci, DAF segregated with the CR1/C4BP segment. Thus, the DAF gene maps closer to the CR1/C4BP loci than to the H gene, from which it can be separated by genetic recombination.

L16 ANSWER 9 OF 9 HCAPLUS COPYRIGHT 1997 ACS
AN 1979:20624 HCAPLUS
DN 90:20624
TI Differences in activation of human and guinea pig complement by retroviruses
AU Bartholomew, Richard M.; Esser, Alfred F.
CS Dep. Mol. Immunol., Scripps Clin. Res. Inst., La Jolla, Calif., USA
SO J. Immunol. (1978), 121(5), 1748-51
CODEN: JOIMA3; ISSN: 0022-1767
DT Journal
LA English
AB C type murine leukemia viruses (retroviruses) have been shown previously to possess a receptor for human C1 that activated human but not guinea pig complement. The present study provides evidence that the viral receptor also binds guinea pig C1 but that such binding does not lead to activation. However, incorporation of human C1s into guinea pig C1 to form a C1 ***hybrid*** results in activation of that ***hybrid*** and in viral lysis. In contrast, incorporation of guinea pig C1s into human C1 abolishes activation by the virus. Thus, C1s governs the activation of C1 of the viral receptor.

=> d his

(FILE 'HOME' ENTERED AT 15:29:58 ON 09 JAN 97)

FILE 'MEDLINE, HCAPLUS' ENTERED AT 15:30:10 ON 09 JAN 97

L1 124 FILE MEDLINE
L2 147 FILE HCAPLUS
TOTAL FOR ALL FILES
L3 271 S REGULAT? (2A) COMPLEMENT (2A) ACTIVAT?
L4 129935 FILE MEDLINE
L5 149124 FILE HCAPLUS
TOTAL FOR ALL FILES
L6 279059 S CHIMER? OR HYBRID?
L7 15 FILE MEDLINE
L8 17 FILE HCAPLUS
TOTAL FOR ALL FILES
L9 32 S L3 AND L6
L10 11 FILE MEDLINE
L11 10 FILE HCAPLUS
TOTAL FOR ALL FILES
L12 21 S L9 AND PY>1991
L13 4 FILE MEDLINE
L14 7 FILE HCAPLUS
TOTAL FOR ALL FILES
L15 11 S L9 NOT L12
L16 9 DUP REM L15 (2 DUPLICATES REMOVED)

=> s (complement receptor 1) or (complement receptor 2) or (decay accelerating factor) or (membrane cofactor protein) or (c4# binding protein) or (factor H)
L17 1499 FILE MEDLINE
L18 1484 FILE HCAPLUS

TOTAL FOR ALL FILES
L19 2983 (COMPLEMENT RECEPTOR 1) OR (COMPLEMENT RECEPTOR 2) OR (DEC
AY ACCELERATING FACTOR) OR (MEMBRANE COFACTOR PROTEIN) OR
(C4# BINDING PROTEIN) OR (FACTOR H)

=> s l19 and l6
L20 85 FILE MEDLINE
L21 106 FILE HCAPLUS

TOTAL FOR ALL FILES
L22 191 L19 AND L6

=> s l22 and py>1991
L23 51 FILE MEDLINE
L24 60 FILE HCAPLUS

TOTAL FOR ALL FILES
L25 111 L22 AND PY>1991

=> s l21 not l25
L26 34 FILE MEDLINE
L27 46 FILE HCAPLUS

TOTAL FOR ALL FILES
L28 80 L21 NOT L25

=> s l28 not l15
L29 30 FILE MEDLINE
L30 43 FILE HCAPLUS

TOTAL FOR ALL FILES
L31 73 L28 NOT L15

=> dup rem l31
PROCESSING COMPLETED FOR L31
L32 51 DUP REM L31 (22 DUPLICATES REMOVED)

=> d bib ab 1-

L32 ANSWER 1 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:229627 HCAPLUS
DN 116:229627
TI Cloning of human cDNA for CD46 isoforms and use of anti-CD46 antibodies or CD46-related nucleic acids in therapy and diagnosis
IN Purcell, Damien Francis John; Russell, Sarah May; McKenzie, Ian Farquar Campbell
PA University of Melbourne, Australia
SO PCT Int. Appl., 77 pp.
CODEN: PIXXD2
PI WO 9118097 A1 911128
DS W: AU, CA, JP, US
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE
AI WO 91-AU199 910510
PRAI AU 90-133 900511
DT Patent
LA English
AB The cDNAs for human CD46 isoforms are cloned. The CD46 proteins may be used to inhibit complement activation or inflammation-mediated immunoresponse (no data); the anti-CD46 antibodies may be used to diagnose or treat leukemia (no data); and CD46-related nucleic acids may be used to treat leukemia or diagnose the likelihood of spontaneous abortion (no data). Fourteen different human CD46 cDNAs were identified. The mRNAs from which these cDNAs were derived were formed by differential splicing and differed primarily in the C-terminal-encoding sequences. Two allelic forms, .alpha. and .beta., of mol. wt. 66 and 56 kDa, resp.; and 4 other isoforms, .gamma., .delta., .epsilon., and .phi. with mol. wts. of 76, 35, 63, and 78 kDa, resp. were studied. Malignant tissue samples and cell lines contained 10-20-fold more CD46 than corresponding non-malignant cells.

L32 ANSWER 2 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:57365 HCAPLUS
DN 116:57365
TI Soluble recombinant ligand-binding protein-Ig fusion proteins with improved serum clearance rates and their therapeutic use
IN Hebell, Thomas; Fearon, Douglas T.
PA Johns Hopkins University, USA
SO PCT Int. Appl., 67 pp.
CODEN: PIXXD2
PI WO 9116437 A1 911031
DS W: AU, CA, DE, JP, KR, US
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE
AI WO 91-US2852 910425
PRAI US 90-513299 900425
DT Patent
LA English
AB Ligand-binding proteins fused to Ig chains are produced with recombinant cells. The ***chimeric*** ligand-binding proteins have decreased serum clearance rates relative to the ligand-binding protein alone. These fusion proteins can be used to treat immune disorders, thrombotic conditions, and myocardial infarcts (no data). The cDNA for the C3dg-binding and Epstein-Barr virus (EBV)-binding short consensus repeats of ***complement*** ***receptor*** ***2*** was fused with the cDNA for Ig .gamma.-1 chain to prep. plasmid pSNRCR2. This ***chimeric*** gene was expressed in J558L myeloma cells and the recombinant CR2-IgG1 fusion protein was purified by affinity chromatog. This protein inhibited EBV infection of RAMOS cells in a dose-dependent manner, and suppressed complement-dependent antibody responses to T-cell-dependent and T-cell-independent antibody responses in a murine model. The initial rate of clearance of this contruct was greater than the Ig alone, but between 10-20 h, the rates were similar. A CR1-F(ab')2 ***chimera*** was also produced and tested.

L32 ANSWER 3 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:52936 HCAPLUS
DN 116:52936
TI ***C4*** ***binding*** ***protein*** fusions with therapeutically useful proteins
IN Pasek, Mark P.; Winkler, Gunther; Liu, Theresa R.
PA Biogen, Inc., USA
SO PCT Int. Appl., 105 pp.
CODEN: PIXXD2
PI WO 9111461 A1 910808
DS W: AU, CA, JP, US
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LU, NL, SE
AI WO 91-US567 910128
PRAI US 90-470888 900126
DT Patent
LA English
AB The complement ***C4*** ***binding*** ***protein*** (C4bp) is used in fusion proteins with therapeutic proteins to ensure targetting of the fusion protein to the blood and to prolong serum half-life. In particular, the short consensus repeats of the N-terminal region are used. The primary aim is to provide CD4 antigens for use in the treatment of AIDS. A cDNA for C4bp was cloned by polymerase chain reaction amplification of the mRNA and subloned into the animal expression vector pJOD-10. When the cloned gene was expressed in COS-7 cells a heptameric C4bp of the correct conformation but lacking the S protein-binding subunit was produced. ***Chimeric*** genes based upon this cDNA and one encoding sol. CD4 antigen were constructed and expressed in COS-7 cells. The purified fusion proteins were shown to form multimers and to bind the glycoprotein gp120 of HIV in vitro. Conditioned medium from producer cells prevented syncytia formation by HIV-infected cells.

L32 ANSWER 4 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1991:556956 HCAPLUS
DN 115:156956
TI Purification of human ***membrane*** ***cofactor*** ***protein*** (MCP), recombinant production of MCP, and therapeutic and diagnostic uses of MCP
IN Atkinson, John P.
PA Washington University, USA
SO PCT Int. Appl., 42 pp.
CODEN: PIXXD2
PI WO 9102002 A1 910221
DS W: AT, AU, BB, BG, BR, CA, CH, DE, DK, ES, FI, GB, HU, JP, KP, KR, LK, LU, MC, MG, MW, NL, NO, RO, SD, SE, SU
RW: AT, BE, BF, BJ, CF, CG, CH, CM, DE, DK, ES, FR, GA, GB, IT, LU, ML, MR, NL, SE, SN, TD, TG
AI WO 90-US4107 900720
PRAI US 89-384210 890721
US 90-510709 900419
DT Patent
LA English

AB Human MCP, a protein involved in regulation of complement activity, has been purified to homogeneity. The cDNAs encoding 6 isoforms of this protein have been retrieved and permit deduction of the complete amino acid sequences and the recombinant prodn. of proteins with this activity. Pharmaceutical compns. in which MCP is the active ingredient for use in treating autoimmune diseases, antibody prepns. for diagnosis, and DNA probes are also disclosed.

L32 ANSWER 5 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1991:466112 HCAPLUS
DN 115:66112
TI Expression of a liver fatty acid binding protein/human ***decay*** - ***accelerating*** ***factor*** /HLA-B44 ***chimeric*** gene in transgenic mice
AU Hansbrough, J. Randall; Lublin, Douglas M.; Roth, Kevin A.; Birkenmeier, Edward A.; Gordon, Jeffrey I.
CS Sch. Med., Washington Univ., St. Louis, MO, 63110, USA
SO Am. J. Physiol. (1991), 260(6, Pt. 1), G929-G939
CODEN: AJPHAP; ISSN: 0002-9513
DT Journal
LA English
AB The intestinal epithelium is characterized by the rapid and continuous renewal of its four principal cell types and by its ability to establish and maintain remarkably complex spatial differentiation along its crypt-to-villus and duodenal-to-colonic axes. The authors previously used transgenic mice contg. liver fatty acid binding protein/human growth hormone (L-FABP/hGH) fusion genes to analyze the mol. mechanisms responsible for encoding positional information in this epithelium. Because these studies could not distinguish whether cis-acting sequences in the L-FABP promoter or hGH structural gene were responsible for the obsd. cellular and regional patterns of transgene transcription in the gut, a second model fusion gene has now been constructed. It consists of nucleotides -596 to +21 of rat L-FABP linked to a cDNA encoding a ***chimeric*** protein, human ***decay*** - ***accelerating*** ***factor*** (DAF, minus the site of attachment of its COOH-terminal glycophospholipid anchor), coupled to the transmembrane (TM) and cytoplasmic domains of human HLA-B44. RNA blot ***hybridization*** and immunocytochem. analyses revealed that the cell-specific and region-specific expressions of DAF-TM and hGH in adult mice appear identical along both axes of the gut, indicating that cis-acting elements contained within the 5' nontranscribed region of the L-FABP gene rather than in the reporter are largely responsible for these obsd. patterns of transgene expression. Unlike pre-hGH, a prototypical secreted protein, DAF-TM is a membrane protein. The ability to direct its expression along the length of both axes of the gut provides an opportunity to analyze in vivo the sorting pathways of membrane-assocd. proteins in normal epithelial cells as a function of their location and differentiation. Light microscopic studies indicate that DAF-TM is targeted to the basolateral and apical surfaces of villus-assocd. enterocytes.

L32 ANSWER 6 OF 51 MEDLINE DUPLICATE 1
AN 91286227 MEDLINE
TI Analysis of Epstein-Barr virus-binding sites on ***complement*** ***receptor*** ***2*** (CR2/CD21) using human-mouse ***chimeras*** and peptides. At least two distinct sites are necessary for ligand-receptor interaction.
AU Molina H; Brenner C; Jacobi S; Gorka J; Carel J C; Kinoshita T; Holers V M
CS Howard Hughes Medical Institute Laboratories, Washington University School of Medicine, St. Louis, Missouri 63110..
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1991 Jul 5) 266 (19) 12173-9.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9110
AB The predicted amino acid sequence of human ***complement*** ***receptor*** ***2*** (CR2, CD21, C3d,g/Epstein-Barr virus receptor) and its genetic murine homologue are approximately 70% identical. The sequence of each consists of a linear array of 60-70 amino acid repeats designated short consensus repeats (SCRs). Although they share significant sequence identity, a major difference in the activities of these two proteins has been believed to be the ability of human, but not mouse, CR2 to mediate Epstein-Barr virus (EBV) infection of B lymphocytes. In order to formally address this question and to directly compare the activities of the CR2 protein of each species, we have expressed recombinant mouse CR2 (rMCR2) in a human K562 erythroleukemia cell line background. We have found that rMCR2 reacts with two previously described rat anti-MCR2 monoclonal antibodies (mAbs), 7G6 and 7E9, but not mAb 8C12, which recognizes only mouse ***complement*** ***receptor*** ***1*** . rMCR2 rosettes with erythrocytes bearing mouse and human C3d,g and binds glutaraldehyde cross-linked human C3d,g with a similar Kd as human CR2 (HCR2). rMCR2 does not bind EBV. By using this observation and constructing ***chimeras*** bearing portions of MCR2 on a HCR2 background, we have been able to define unique sequences in HCR2 SCRs 1 and 2 important in the interaction with both mAb OKB7, which blocks EBV binding and infection, and with EBV. In addition, by using blocking peptides derived from HCR2 sequence, we have identified a second distinct region in SCR2 important in EBV binding. Therefore, within the first two SCRs of HCR2 are multiple distinct sites of interaction with EBV and with mAb OKB7.

L32 ANSWER 7 OF 51 MEDLINE
AN 91268081 MEDLINE
TI Molecular cloning of a human serum protein structurally related to complement ***factor*** ***H*** .
AU Skerka C; Horstmann R D; Zipfel P F
CS Bernhard Nocht Institute for Tropical Medicine, Hamburg, Federal Republic of Germany..
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1991 Jun 25) 266 (18) 12015-20.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK-X56209; GENBANK-X56210
EM 9109
AB Two cDNA clones termed H36-1 and H36-2 were isolated from a human liver cDNA library. Clone H36-1 appears to represent the recently isolated human serum proteins h37 and h42, which are two differently glycosylated forms of a protein antigenically related to human complement ***factor*** ***H*** . The H36-1 deduced protein sequence is 327 amino acid long and possesses a leader sequence. The secreted part of the protein is comprised of five tandem repeating units, termed short consensus repeats (SCRs). SCR 1 and 2 display high homology to the corresponding region of the recently isolated murine ***factor*** ***H*** -related cDNA clone 13G1. In contrast, the 3'-end of the H36-1 clone shows sequence homology to the 3'-end of human complement ***factor*** ***H*** . The second clone, H36-2, is nearly identical to H36-1. Within 1148 base pairs, where the two clones overlap, their nucleotide sequences differed at nine positions. One nucleotide exchange in the sequence of H36-2 which was located within SCR 1 creats a stop codon (TAA). Consequently, the corresponding mRNA cannot code for a functional protein, suggesting that this clone is a transcribed pseudogene. These two clones represent new human members of the family of proteins structurally related to complement ***factor*** ***H*** .

L32 ANSWER 8 OF 51 MEDLINE DUPLICATE 2
AN 91237847 MEDLINE
TI Human immunodeficiency virus infection and syncytium formation in HeLa cells expressing glycophospholipid-anchored CD4.
AU Kost T A; Kessler J A; Patel I R; Gray J G; Overton L K; Carter S G
CS Department of Molecular Biology, Glaxo Research Institute, Research Triangle Park, North Carolina 27709..
SO JOURNAL OF VIROLOGY, (1991 Jun) 65 (6) 3276-83.
Journal code: KCV. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9108
AB The CD4 molecule, a glycoprotein expressed primarily on the cell surface of specific T lymphocytes, is thought to function in T-cell antigen recognition and activation. In addition, CD4 serves as a receptor for human immunodeficiency virus type 1 (HIV-1) by a direct interaction with the HIV-1 surface glycoprotein (gp120). To further characterize the HIV-1-cell interaction, a HeLa cell line was established that expressed a ***chimeric*** molecule of CD4 and ***decay*** - ***accelerating*** ***factor*** (DAF). In the ***chimeric*** CD4-DAF molecule the transmembrane and cytoplasmic domains of CD4 were deleted and replaced with the carboxy-terminal 37 amino acids of DAF. This resulted in the anchoring of the extracellular domain of CD4 to the cell membrane via a glycophospholipid linkage. The glycophosphotipid-anchored CD4 had a molecular size of approximately 56 to 62 kDa and was released following treatment of the cells with phosphatidylinositol-specific phospholipase C. HeLa cells expressing the CD4-DAF ***hybrid*** could be infected with HIV-1, as evidenced by reverse transcriptase activity, p24 core antigen content, and infectious virus production. In addition, transfection of the HeLa CD4-DAF cells with a plasmid that directs the synthesis of HIV-1 envelope glycoproteins or cocultivation with HeLa cells expressing the virus glycoproteins resulted in syncytium formation. These results indicate that the transmembrane and cytoplasmic domains of the CD4 molecule are dispensable for both HIV infection and syncytium formation.

L32 ANSWER 9 OF 51 MEDLINE DUPLICATE 3
AN 91201892 MEDLINE
TI Cloning of the 1.4-kb mRNA species of human complement ***factor*** ***H*** reveals a novel member of the short consensus repeat family related to the carboxy terminal of the classical 150-kDa molecule.
AU Estaller C; Koistinen V; Schwaeble W; Dierich M P; Weiss E H
CS Institut fur Immunologie, Munchen, FRG..
SO JOURNAL OF IMMUNOLOGY, (1991 May 1) 146 (9) 3190-6.
Journal code: IFB. ISSN: 0022-1767.
CY United States

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 9107
AB Three ***factor*** ***H*** mRNA species of 4.3 kb, 1.8 kb, and 1.4 kb are constitutively expressed in human liver. Having previously characterized full-length cDNA clones derived from the 4.3-kb and 1.8-kb factor mRNA, we report here the isolation and eucaryotic expression of full-length cDNA clones coding for the 1.4-kb mRNA species. The 1266-bp cDNA codes for a polypeptide of 330 amino acids and contains two polyadenylation signals and a short poly(A)+tail. The protein is composed of a leader peptide followed by five short consensus repeat domains. It shows a ***hybrid*** structure with the last three domains being almost identical to the carboxy-terminal of the classical 150-kDa ***factor*** ***H*** molecule and the two first domains representing unique short consensus repeat structures. Eucaryotic expression in COS7 cells revealed two polypeptides derived from one cDNA clone that are also found in human serum. Differences between the cDNA clones within the last three domains indicate two distinct, possibly allelic sequences that, in addition, differ from the authentic 150-kDa ***factor*** ***H*** sequence. Southern blot results support the notion that the 4.3-kb ***factor*** ***H*** and the 1.4-kb ***factor*** ***H*** -related mRNA are transcribed from two separate but highly homologous genes.

L32 ANSWER 10 OF 51 MEDLINE DUPLICATE 4
AN 91364778 MEDLINE
TI Structure-function studies on human ***C4b*** - ***binding*** ***protein*** using monoclonal antibodies.
AU Hessing M; Kanters D; Heijnen H F; Hackeng T M; Sixma J J; Bouma B N
CS Department of Haematology, University Hospital Utrecht, The Netherlands..
SO EUROPEAN JOURNAL OF IMMUNOLOGY, (1991 Sep) 21 (9) 2077-85.
Journal code: EN5. ISSN: 0014-2980.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9112
AB Human ***C4b*** - ***binding*** ***protein*** (C4BP) is a multimeric regulatory complement component interacting with vitamin K-dependent protein S and complement C4b. Using ***hybridoma*** technology, a panel of monoclonal antibodies (mAb) specific for intact human C4BP and its 160-kDa chymotryptic central core fragment were prepared to study the structure-function relationships of C4BP. By Western blot analysis and competition experiments, four distinct groups of mAb were identified and mapped on the C4BP molecule. By rotary shadowing, spider-like images of C4BP-antibody complexes were obtained and immunoelectron microscopy provided some information on the stoichiometry of the antibody-C4BP interaction. Certain antibodies interacted with C4BP molecules only at a ratio of 1:1. Others formed complexes of two or more antibodies bound to homologous sites on the C4BP molecule. Using an enzyme-linked immunosorbent sandwich assay for the measurement of the complex formation between protein S and C4BP, mAb against the central core and the disulfide-linked beta chain of C4BP were identified that inhibited the binding of protein S to C4BP. In a binding assay using 125I-labeled C4BP and solid-phase C4b, the inhibitory effect of one group of anti-C4BP mAb on the binding of C4BP to C4b was demonstrated.

L32 ANSWER 11 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1991:630035 HCAPLUS
DN 115:230035
TI Determination of the structural basis for selective binding of Epstein-Barr virus to human complement receptor type 2
AU Martin, Don R.; Yuryev, Anton; Kalli, Kimberly R.; Fearon, Douglas T.; Ahearn, Joseph M.
CS Sch. Med., Johns Hopkins Univ., Baltimore, MD, 21205, USA
SO J. Exp. Med. (1991), 174(6), 1299-311
CODEN: JEMEAV; ISSN: 0022-1007
DT Journal
LA English
AB Epstein-Barr virus (EBV) is an oncogenic herpesvirus that selectively infects and immortalizes human B lymphocytes. One determinant of this narrow tropism is human CR2, the only viral receptor within the superfamily of proteins that contain short consensus repeats (SCRs). Human CR2 serves as a receptor for both C3dg and the gp350/220 glycoprotein of EBV, and binds the monoclonal antibody (mAb), OKB7, which blocks binding of both ligands to the receptor. In contrast, although murine CR2 is capable of binding human C3dg and this interaction can be blocked with the mAb 7G6, it does not bind OKB7 or EBV. The structural basis was detd. for abs. specificity of EBV for human CR2 through characterization of a panel of 24 human-murine ***chimeric*** receptors, all of which bind human C3dg. The results indicate that perferential binding of EBV to human CR2 is not due to unique amino acids that are capable of binding the virus, but reflects a distinct receptor conformation that can be achieved in murine CR2 with single amino acid substitutions in two discontinuous regions of the primary structure: replacement of proline at position 15 with the corresponding serine from human CR2, and elimination of a potential N-linked glycosylation site between SCR-1 and SCR-2. Furthermore, species-specific binding of EBV, OKB7, and 7G6 can all be manipulated through substitutions among residues 8-15, suggesting that this octapeptide is part of a structural determinant that is crit. for binding of both viral and natural ligands to CR2.

L32 ANSWER 12 OF 51 MEDLINE
AN 91281861 MEDLINE
TI Expression of a liver fatty acid binding protein/human ***decay*** - ***accelerating*** ***factor*** /HLA-B44 ***chimeric*** gene in transgenic mice.
AU Hansbrough J R; Lublin D M; Roth K A; Birkenmeier E A; Gordon J I
CS Departments of Medicine, Washington University School of Medicine, St. Louis, Missouri 63110..
NC DK-30292 (NIDDK)
SO AMERICAN JOURNAL OF PHYSIOLOGY, (1991 Jun) 260 (6 Pt 1) G929-39.
Journal code: 3U8. ISSN: 0002-9513.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9110
AB The intestinal epithelium is characterized by the rapid and continuous renewal of its four principal cell types and by its ability to establish and maintain remarkably complex spatial differentiation along its crypt-to-villus and duodenal-to-colonic axes. We have previously used transgenic mice containing liver fatty acid binding protein/human growth hormone (L-FABP/hGH) fusion genes to analyze the molecular mechanisms responsible for encoding positional information in this epithelium. Because these studies could not distinguish whether cis-acting sequences in the L-FABP promoter or hGH structural gene were responsible for the observed cellular and regional patterns of transgene transcription in the gut, a second model fusion gene has now been constructed. It consists of nucleotides -596 to +21 of rat L-FABP linked to a cDNA encoding a ***chimeric*** protein, human ***decay*** - ***accelerating*** ***factor*** (DAF, minus the site of attachment of its COOH-terminal glycophospholipid anchor), coupled to the transmembrane (TM) and cytoplasmic domains of human HLA-B44. RNA blot ***hybridization*** and immunocytochemical analyses revealed that the cell-specific and region-specific expressions of DAF-TM and hGH in adult mice appear identical along both axes of the gut, indicating that cis-acting elements contained within the 5' nontranscribed region of the L-FABP gene rather than in the reporter are largely responsible for these observed patterns of transgene expression. Unlike pre-hGH, a prototypical secreted protein, DAF-TM is a membrane protein. The ability to direct its expression along the length of both axes of the gut provides an opportunity to analyze in vivo the sorting pathways of membrane-associated proteins in normal epithelial cells as a function of their location and differentiation. Light microscopic studies indicate that DAF-TM is targeted to the basolateral and apical surfaces of villus-associated enterocytes.

L32 ANSWER 13 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:100335 HCAPLUS
DN 116:100335
TI Cloning and expression of human acidic fibroblast growth ***factor*** (***h*** .alpha.FGF) in Escherichia coli
AU Wang, Haoyong; Wang, Huixin; Huang, Peitang; Xiue, Yianning; Li, Fengsheng; Rui, Xianliang; Zhao, Qiang; Zhou, Tingchong
CS Inst. Basic Med. Sci., Acad. Mil. Med. Sci., Beijing, 100850, Peop. Rep. China
SO Shengwu Huaxue Zazhi (1991), 7(6), 713-18
CODEN: SHZAE4; ISSN: 1000-8543
DT Journal
LA Chinese
AB The human acidic fibroblast growth ***factor*** (***h*** .alpha.FGF) gene was inserted into the EcoRI site of plasmid pBV220. The recombinant plasmids were identified by in situ colony ***hybridization*** , enzyme digestion and Southern blot ***hybridization*** . The plasmid pBV220-.alpha.FGF showed the h.alpha.FGF gene located immediately after the PLPR promoter in the right direction. Bacteria that contain the plamid pBV-.alpha.FGF was treated by 42.degree.C heat induction, and its lysates stimulated DNA synthesis if 3T3 cell. SDS-PAGE showed that the haFGE gene expression product is about 15% of the total bacteria proteins.

L32 ANSWER 14 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1991:119939 HCAPLUS
DN 114:119939
TI Proposed structure of the F' allotype of human CR1. Loss of a C3b binding site may be associated with altered function
AU Wong, Winnie W.; Farrell, Scott A.
CS Dep. Rheumatol. Immunol., Brigham and Women's Hosp., Boston, MA, 02115, USA
SO J. Immunol. (1991), 146(2), 656-62
CODEN: JOIMA3; ISSN: 0022-1767
DT Journal
LA English
AB Human CR1 is composed of tandem long homologous repeating (LHR)

segments that encode sep. binding sites for C3b or C4b. Homologous recombination with unequal crossover has been proposed as the genetic mechanism that gave rise to the CR1 alleles that differed in their total nos. of LHR. The F allotype has four LHR, named LHR-A, -B, -C, -D, 5' to 3'. The site in LHR-A preferentially binds C4b and those in LHR-B and -C prefer C3b. A previous study revealed the presence of a 5th LHR with sequences similar to LHR-B and a 3rd C3b binding site in the S allotype of higher mol. wt. In the present study, an 18-kb EcoRV fragment that was assocd. with the expression of the lower mol. wt. F' allotype ***hybridized*** with a unique pattern of cDNA and intron probes specific for LHR-C. Deletion of LHR-B and one C3b binding site was proposed as the mechanism for the appearance of this F'-specific fragment. Functional differences among the CR1 variants were sought by comparative analyses of sol. rCR1 having 1, 2, or 3 C3b binding sites. Although these 3 variants did not exhibit any significant differences in their capacities to act as cofactors for the cleavage of monomeric C3b, their relative affinities for dimeric ligand varied >100-fold. Furthermore, the variant with only one C3b binding site was at least 10-fold less effective in the inhibition of the alternative pathway C3 and C5 convertases. Thus, the F' allotype may be impaired in its capacity to bind opsonized immune complexes, to inhibit the formation of the alternative pathway C3 and C5 convertases, and perhaps to mediate other CR1-dependent cellular responses.

L32 ANSWER 15 OF 51 MEDLINE DUPLICATE 5
AN 92126004 MEDLINE
TI Chromosome assignments of the genes for glucocorticoid receptor, myelin basic protein, leukocyte common antigen, and TRPM2 in the rat.
AU Goldner-Sauve A; Szpirer C; Szpirer J; Levan G; Gasser D L
CS Department of Human Genetics, University of Pennsylvania School of Medicine, Philadelphia 19104..
NC P50 DE09164 (NIDR)
SO BIOCHEMICAL GENETICS, (1991 Jun) 29 (5-6) 275-86.
Journal code: 9YK. ISSN: 0006-2928.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9204
AB We have utilized rat-mouse somatic cell ***hybrids*** to make chromosomal assignments for the glucocorticoid receptor (GR), myelin basic protein (MBP), leukocyte common antigen (LCA), and testosterone-repressed prostate message-2 (TRPM2) genes in the rat. The genes for GR and MBP both map on chromosome 18 of the rat, which corresponds to the mapping of both genes on chromosome 18 of the mouse. The gene for LCA maps on chromosome 13, which is where ***C4b*** - ***binding*** ***protein*** beta-chain (C4BPB), coagulation factor V (F5), and renin have previously been assigned. This linkage group appears to be homologous to a substantial portion of mouse chromosome 1 and human chromosome 1q. Finally, the TRPM2 gene has been assigned to rat chromosome 15.

L32 ANSWER 16 OF 51 MEDLINE
AN 92079888 MEDLINE
TI Molecular cloning and nucleotide sequence of the glycogen branching enzyme gene (glgB) from Bacillus stearothermophilus and expression in Escherichia coli and Bacillus subtilis.
AU Kiel J A; Boels J M; Beldman G; Venema G
CS Department of Genetics, Center of Biological Sciences, The Netherlands..
SO MOLECULAR AND GENERAL GENETICS, (1991 Nov) 230 (1-2) 136-44.
Journal code: NGP. ISSN: 0026-8925.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-M35089; GENBANK-S69712; GENBANK-Z14057; GENBANK-S63909;
GENBANK-S55527; GENBANK-S55528; GENBANK-S55529; GENBANK-S55530;
GENBANK-S55531; GENBANK-S55532
EM 9203
AB The structural gene for the Bacillus stearothermophilus glycogen branching enzyme (glgB) was cloned in Escherichia coli. Nucleotide sequence analysis revealed a 1917 nucleotide open reading frame (ORF) encoding a protein with an Mr of 74787 showing extensive similarity to other bacterial branching enzymes, but with a shorter N-terminal region. A second ORF of 951 nucleotides encoding a 36971 Da protein started upstream of the glgB gene. The N-terminus of the ORF2 gene product had similarity to the Alcaligenes eutrophus czcD gene, which is involved in cobalt-zinc-cadmium resistance. The B. stearothermophilus glgB gene was preceded by a sequence with extensive similarity to promoters recognized by Bacillus subtilis RNA polymerase containing sigma ***factor*** ***H*** (E - sigma H). The glgB promoter was utilized in B. subtilis exclusively in the stationary phase, and only transcribed at low levels in B. subtilis spoOH, indicating that sigma ***factor*** ***H*** was essential for the expression of the glgB gene in B. subtilis. In an expression vector, the B. stearothermophilus glgB gene directed the synthesis of a thermostable branching enzyme in E. coli as well as in B. subtilis, with optimal branching activity at 53 degrees C.

L32 ANSWER 17 OF 51 MEDLINE DUPLICATE 6
AN 91177965 MEDLINE
TI An internally positioned signal can direct attachment of a glycophospholipid membrane anchor.
AU Caras I W
CS Department of Immunobiology, Genentech, Inc., South San Francisco, California 94080..
SO JOURNAL OF CELL BIOLOGY, (1991 Apr) 113 (1) 77-85.
Journal code: HMV. ISSN: 0021-9525.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9107
AB All known glycophosphatidylinositol (GPI)-anchored membrane proteins contain a COOH-terminal hydrophobic domain necessary for signalling anchor attachment. To examine the requirement that this signal be at the COOH terminus of the protein, we constructed a ***chimeric*** protein, DAFhGH, in which human growth hormone (hGH) was fused to the COOH terminus of ***decay*** ***accelerating*** ***factor*** (DAF) (a GPI-anchored protein), thereby placing the GPI signal in the middle of the ***chimeric*** protein. We show that the fusion protein appears to be processed at the normal DAF processing site in COS cells, producing GPI-anchored DAF on the cell surface. This result indicates that the GPI signal does not have to be at the COOH terminus to direct anchor addition, suggesting that the absence of a hydrophilic COOH-terminal extension (beyond the hydrophobic domain) is not a necessary requirement for GPI anchoring. A similar DAFhGH fusion, containing an internal GPI signal in which the DAF hydrophobic domain was replaced with the signal peptide of hGH, also produced GPI-anchored cell surface DAF. The signal for GPI attachment thus exhibits neither position specificity nor sequence specificity. In addition, mutant DAF or DAFhGH constructs lacking an NH2-terminal signal peptide failed to produce GPI-anchored protein, suggesting that membrane translocation is necessary for anchor addition.

L32 ANSWER 18 OF 51 MEDLINE DUPLICATE 7
AN 90153969 MEDLINE
TI Identification and sequence analysis of four complement ***factor*** ***H*** -related transcripts in mouse liver.
AU Vik D P; Munoz-Canoves P; Kozono H; Martin L G; Tack B F; Chaplin D D
CS Department of Immunology, Research Institute of Scripps Clinic, La Jolla, California 92037..
NC AI 17354 (NIAID)
AI 07706 (NIAID)
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1990 Feb 25) 265 (6) 3193-201.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK-J05259
EM 9005
AB Mouse liver RNA analyzed by Northern blotting with a full-length complement ***factor*** ***H*** cDNA probe demonstrates the 4.4-kilobase (kb) H mRNA as well as three additional ***hybridizing*** species of 3.5, 2.8, and 1.8 kb, respectively. Further characterization of these alternative transcripts was pursued by isolation of additional cDNAs from a liver library using a full-length H probe. Twelve clones homologous to but distinct from H were isolated, analyzed by restriction mapping, and divided into four classes, A, B, C, and D, based on their sequences. Clones from classes A, B, and C all contained nearly identical 5'-untranslated regions and leader sequences that differed from H at more than 50% of their nucleotide positions. The 5'-untranslated and leader sequences of the class D clone were unrelated to the corresponding regions of H or the class A, B, or C clones. The remaining portions of the H-related cDNAs were made up of short consensus repeats, 7 in class A, 4 in class B, 13 in class C, and 5 in class D. To determine the relationship between the H-related transcripts and the cDNA clones, Northern blots of liver RNA were analyzed by ***hybridization*** with two probes, one specific for the class D cDNA and the other reacting specifically with the class A, B, and C cDNAs. The class A/B/C probe detected transcripts of 3.5, 2.8, and 1.8 kb in liver RNA, and the class D probe ***hybridized*** to a distinct 1.8-kb message. Additionally, a cosmid genomic library was screened with H cDNA, and nine H-related clones were isolated. They spanned a region of approximately 120 kb, defining at least two discrete H-related gene loci. These results identify new members of the super-family of C3b/ ***C4b*** ***binding*** ***protein*** genes.

L32 ANSWER 19 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1990:530285 HCAPLUS
DN 113:130285
TI Expression of T cell antigen receptor heterodimers in a lipid-linked form
AU Lin, Augustine Y.; Devaux, Brigitte; Green, Adrienne; Sagerstrom,

Charles; Elliott, John F.; Davis, Mark M.
CS Sci. Med., Stanford Univ., Stanford, CA, 94305-5402, USA
SO Science (Washington, D. C., 1883-) (1990), 249(4969), 677-9
CODEN: SCIEAS; ISSN: 0036-8075
DT Journal
LA English
AB The interaction of the T cell receptor for antigen (TCR) with its antigen-major histocompatibility complex ligand is difficult to study because both are cell surface multimers. The TCR consists of two chains (.alpha. and .beta.) that are complexed to the five or more nonpolymorphic CD3 polypeptides. A sol. form of the TCR was engineered by replacing the carboxyl termini of .alpha. and .beta. with signal sequences from lipid-linked proteins, making them susceptible to enzymic cleavage. In this manner, TCR heterodimers can be expressed independently of the CD3 polypeptides and in significant quantities (0.5 mg per wk). This technique seems generalizable to biochem. and structural studies of many other cell surface mols. as well.

L32 ANSWER 20 OF 51 MEDLINE
AN 90111033 MEDLINE
TI Demonstration of an unusual allelic variation of mouse ***factor*** ***H*** by the complete cDNA sequence of the H.2 allotype.
AU Natsuume-Sakai S; Nonaka M; Nonaka M; Harada Y; Shreffler D C; Moriwaki K
CS Department of Immunobiology, Kanazawa University, Japan..
SO JOURNAL OF IMMUNOLOGY, (1990 Jan 1) 144 (1) 358-62.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
OS GENBANK-M31979; GENBANK-M31980
EM 9004
AB Three allotypes of murine ***factor*** ***H*** have been identified serologically in the previous study (denoted H.1, H.2, and H.3). A cDNA clone coding for the entire length of murine ***factor*** ***H*** was isolated from a library constructed from the livers of STR/N mice which have H.2 allotype and was fully sequenced. The insert of this clone (STR309) contained 4184 nucleotides and consisted of a 47-bp 5' noncoding region, a 54-bp coding for leader peptide, a 3648 bp for the mature ***factor*** ***H*** protein, and a 435-bp 3' noncoding region. Compared with the previously reported sequence of the cDNA clone (MH8) isolated from B10.WR mice that have H.1 allotype, the size of the protein coding region was exactly the same, but 21 nucleotide substitutions resulting in 15 amino acid replacements were observed. The amino acid replacement/nucleotide substitution ratio (0.71) is far higher than those observed in the allotypic variations of other proteins. Four 15-base oligonucleotide probes specific for either STR309 or MH8 were synthesized and used in Northern blot analysis. The probes specific for STR309 ***hybridized*** with mRNA isolated from the livers of STR/N mice but not with mRNA from the livers of BALB/c mice that have H.1 allotype, whereas the reverse pattern was observed with the oligonucleotide probes specific for MH8. These results strongly suggest that the nucleotide sequence of STR309 represents H.2 allotype of ***factor*** ***H*** protein, providing an example of an unusual allotype with high ratio of amino acid replacements to nucleotide substitutions.

L32 ANSWER 21 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1990:585798 HCAPLUS
DN 113:185798
TI Expression of CR1 (CD35) mRNA in podocytes from adult and fetal human kidneys
AU Appay, Marie Dominique; Kazatchkine, Michel D.; Levi-Strauss, Matthieu; Hinglais, Nicole; Bariety, Jean
CS Hop. Broussais, Paris, Fr.
SO Kidney Int. (1990), 38(2), 289-93
CODEN: KDYIA5; ISSN: 0085-2538
DT Journal
LA English
AB The presence of CR1 (human ***complement*** ***receptor*** ***1***) mRNA in podocytes was investigated using a 35S-labeled CR1 cDNA probe and in situ ***hybridization*** in sections from fetal and adult human kidneys. CR1 mRNA was only detected in immature podocytes at early stages of glomerular differentiation in the fetal kidney. In contrast, CR1 antigen was abundantly expressed on immature and mature podocytes in fetal kidneys and adult glomeruli. Thus, the expression of the CR1 gene in podocytes is tightly regulated. It is possible that the prolonged life span of adult podocytes is assocd. with a slow turnover of CR1 and low or intermittent accumulation of CR1 mRNA transcripts.

L32 ANSWER 22 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1990:92974 HCAPLUS
DN 112:92974
TI Demonstration of an unusual allelic variation of mouse ***factor*** ***H*** by the complete cDNA sequence of the H.2 allotype
AU Natsuume-Sakai, Shunnosuke; Nonaka, Masaru; Nonaka, Mayumi; Harada, Yoshinobu; Shreffler, Donald C.; Moriwaki, Kazuo
CS Cancer Res. Inst., Kanazawa Univ., Kanazawa, 920, Japan
SO J. Immunol. (1990), 144(1), 258-62
CODEN: JOIMA3; ISSN: 0022-1767
DT Journal
LA English
AB Three allotypes of murine ***factor*** ***H*** have been identified serol. (denoted H.1, H.2, and H.3). A cDNA clone coding for the entire length of murine ***factor*** ***H*** was isolated from a library constructed from the livers of STR/N mice which have the H.2 allotype and was fully sequenced. The insert of this clone (STR309) contained 4184 nucleotides and consisted of a 47-bp 5' noncoding region, a 54-bp coding for leader peptide, a 3648 bp for the mature ***factor*** ***H*** protein, and a 435-bp 3' noncoding region. Compared with the previously reported sequence of the cDNA clone (MH8) isolated from B10.WR mice that have the H.1 allotype, the size of the protein coding region was exactly the same, but 21 nucleotide substitutions resulting in 15 amino acid replacements were obsd. The amino acid replacement/nucleotide substitution ratio (0.71) is far higher than those obsd. in the allotypic variations of other proteins. Four 15-base oligonucleotide probes specific for either STR309 or MH8 were synthesized and used in Northern blot anal. The probes specific for STR309 ***hybridized*** with mRNA isolated from the livers of STR/N mice but not with mRNA from the livers of BALB/c mice that have H.1 allotype, whereas the reverse pattern was obsd. with the oligonucleotide probes specific for MH8. These results strongly suggest that the nucleotide sequence of STR309 represents an H.2 allotype of ***factor*** ***H*** protein, providing an example of an unusual allotype with a high ratio of amino acid replacements to nucleotide substitutions.

L32 ANSWER 23 OF 51 MEDLINE
AN 90125855 MEDLINE
TI ***Factor*** ***H*** .
AU Vik D P; Munoz-Canoves P; Chaplin D D; Tack B F
CS Department of Immunology, Scripps Clinic and Research Foundation, La Jolla, CA 92037..
NC AI19222 (NIAID)
AI17354 (NIAID)
AI07706 (NIAID)
SO CURRENT TOPICS IN MICROBIOLOGY AND IMMUNOLOGY, (1990) 153 147-62.
Ref: 90
Journal code: DWQ. ISSN: 0070-217X.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
EM 9005
AB While the mouse and human H proteins are structurally and functionally similar, they differ in their genetics. Whereas there is no evidence in humans for more than one gene; in mice the H locus is complex. Based on cDNA sequence and ***hybridization*** analysis of genomic cosmid clones, there are at least three distinct genes, all highly related to one another. The consensus repeating unit that comprises this molecule has obviously been duplicated numerous times, since it is present in many other molecules. Thus, it is not surprising to discover that there are several genes related to H in the mouse. A similar case has been described for two other members of this family. In humans, CR1 cDNA ***hybridizes*** to two distinct genomic clusters in the CR1 locus (Wong et al. 1989), and in mice, mCRY ***hybridizes*** to two regions in the genome, one on chromosome 1 and another on chromosome 8 (Aegerter-Shaw et al. 1987). It will be of interest to see if any other members of this family display as complex a genetic locus as murine H.

L32 ANSWER 24 OF 51 MEDLINE DUPLICATE 8
AN 91030518 MEDLINE
TI Application of protein A-rosette assay for screening of monoclonal antibodies to human complement regulatory proteins.
AU Seya T; Hara T; Uenaka A; Nakayama E; Akedo H
CS Department of Immunology, Center for Adult Diseases, Osaka, Japan..
SO COMPLEMENT AND INFLAMMATION, (1990) 7 (2) 78-89.
Journal code: DOQ. ISSN: 1012-8204.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9102
AB Mice were immunized with purified ***membrane*** ***cofactor*** ***protein*** (MCP) and its monoclonal antibodies were screened by protein A(PA)-rosette assay. In this assay, the culture supernatants of ***hybridoma*** cells were layered over fixed MCP-bearing cells, and after washing, PA-coated sheep erythrocytes were applied as an indicator to these MCP-bearing cells. No purified antigen was therefore required throughout the screening. More than 300 of the supernatants harvested were successfully examined within 6 h. Each resultant antibody consisted of a single subclass of IgG, and reacted only with MCP in both transblotted and surface-labeled materials. The sensitivity of this

assay was then assessed with these purified antibodies. As little as 0.5 micrograms of IgG1 or 0.01 micrograms of IgG2a was found to be detectable with more than 30% rosette formation. There were variations among cell lines in the sensitivity to the PA-rosette assay and the sensitivity did not correlate with the quantity of MCP surface expression in any of the cell lines. K562 gave the lowest background (nonspecific rosette formation) and the best specificity for anti-MCP of the 20 MCP-positive cell lines tested. Cell lines suitable for the detection of monoclonal anti- ***decay*** - ***accelerating*** ***factor*** and anti-C3b/C4b receptor were also examined and CCRF-SB and HSB2, and peripheral blood granulocytes, were found to be proper cell lines for screening the ***decay*** - ***accelerating*** ***factor*** and C3b/C4b receptor, respectively. Clones for anti C3b/C4b receptor were successfully obtained using granulocytes by the PA-rosette assay. This method needs no purified antigen and facilitates the rapid screening and purification of positive clones against cell-surface complement regulatory proteins.

L32 ANSWER 25 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1989:548036 HCAPLUS
DN 111:148036
TI Recombinant ***decay*** - ***accelerating*** ***factor*** and a phospholipid anchor domain suitable for membrane binding of fusion proteins
IN Caras, Ingrid W.
PA Genentech, Inc., USA
SO PCT Int. Appl., 61 pp.
CODEN: PIXXD2
PI WO 8901041 A1 890209
DS W: AU, JP
RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE
AI WO 88-US2648 880803
PRAI US 87-83757 870806
DT Patent
LA English
AB The ***decay*** - ***accelerating*** ***factor*** of complement has been cloned and the sequence encoding the phospholipid anchor domain identified. The anchor domain sequence is used to supply a membrane binding sequence for fusion protein. The DNA sequence for the C-terminal 37 amino-acids of ***decay*** - ***accelerating*** ***factor*** were fused to the sequence for the first 300 N-terminal amino-acids of glycoprotein gD1 of herpes simplex virus. This protein is normally secreted into the medium because it lacks an anchor domain. This construct was cloned into a mammalian expression vector and introduced into CHO cells. Immunofluorescence studies showed the fusion protein to be specifically bound to the plasma membrane. Controls showed no inherent binding by the gD-1 sequence to the plasma membrane. The fusion protein was released from the membrane by treatments that removed the covalently bound lipid component of the anchor domain.

L32 ANSWER 26 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1990:95104 HCAPLUS
DN 112:95104
TI Protein S determination in human plasma by EIA using monoclonal antibody
IN Suzuki, Koji; Kyotani, Misako; Mizuki, Kiyoshi; Mikawaya, Kyoko; Shima, Hidekuni; Iwata, Kazushi
PA Fuji Chemical Industry Co., Ltd., Japan
SO Jpn. Kokai Tokkyo Koho, 10 pp.
CODEN: JKXXAF
PI JP 01232264 A2 890918 Heisei
AI JP 88-58439 880314
DT Patent
LA Japanese
AB The title EIA is a sandwich EIA using monoclonal antibody recognizing free protein S and a protein S-complement C 4b-binding protein complex. The monoclonal antibody was prepd. by the conventional ***hybridoma*** method. For total protein S detn. in blood plasma, a sample was incubated at room temp. for 1 h in a monoclonal antibody-sensitized plate, followed by incubation with peroxidase-labeled monoclonal antibody (IgG) and measurement of enzyme activity. For free protein S detn., .gtoreq.1 of the antibodies used recognizes free protein S alone. The method is accurate and specific.

L32 ANSWER 27 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1990:73416 HCAPLUS
DN 112:73416
TI Method, reagent, and kit for immunoassay of human protein S, and monoclonal antibody for use therein
IN Suzuki, Hideaki; Kubota, Takaharu; Hasegawa, Ryoichi; Koike, Yukiya
PA Teijin Ltd., Japan
SO Eur. Pat. Appl., 12 pp.
CODEN: EPXXDW
PI EP 315447 A2 890510
DS R: AT, BE, CH, DE, ES, FR, GB, IT, LI, NL, SE
AI EP 88-310354 881103
PRAI JP 87-279362 871106
DT Patent
LA English
AB The immunol. measurement of human protein S (PS) uses an antibody bound to an insol. carrier and a labeled antibody. One of the antibodies is a polyclonal antibody specifically recognizing human PS; the other antibody is a monoclonal antibody (MAb) which does not recognize the complex of PS with C4bp (high-mol.-wt. ***C4b*** - ***binding*** ***protein***) of the human complement system, but does recognize free human PS. A MAb with the above specificity is produced. A kit for immunol. measurement of human PS which incorporates the above antibodies is described. The method is easily performed and can measure free human PS in a soln. with high sensitivity. Mice were immunized against purified human PS, and ***hybridomas*** were produced, cloned, and selected by std. techniques. The MAb produced from each of 6 selected clones was purified by chromatog. on protein A-Sepharose 4B; relative specific binding capacities of the 6 MAb to the PS-C4bp complex was detd. The MAb 2B9F12, which specifically recognizes free human PS, was labeled with horseradish peroxidase. A com. goat anti-human PS polyclonal antibody was immobilized on polystyrene beads. Purified PS (0-400 ng/mL) was mixed with the above antibodies, and, after incubation, aspiration, and washing, the enzyme activity on the beads was detd. Absorbance at 650 nm was related to PS concn.; the blank (no PS) sample showed background absorbance. In a sep. detn., PS concn. (4.0 and 8.0 .mu.g/mL) was, within the error of the measurement, identical in the absence or presence of C4bp-PS complex.

L32 ANSWER 28 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1989:623846 HCAPLUS
DN 111:223846
TI Structural and magnetization density studies of lanthanum nickel oxide (La2NiO4)
AU Lander, G. H.; Brown, P. J.; Spalek, J.; Honig, J. M.
CS Jt. Res. Cent., Comm. Eur. Communities, Karlsruhe, D-7500, Fed. Rep. Ger.
SO Phys. Rev. B: Condens. Matter (1989), 40(7-A), 4463-71
CODEN: PRBMDO; ISSN: 0163-1829
DT Journal
LA English
AB Neutron diffraction expts. were performed on crystals of LaiNiO4. Above 70 k the structure is orthorhombic Bmab. At 70 K a first-order transition occurs to a crystal structure that may be tetragonal P42/ncm. With resoln., there is obsd. no splitting of the diffraction peaks that would indicate the lower-symmetry orthorhombic Pccn. A crystallog. refinement of 307 independent reflections from 2 different crystals gives an R factor of 6% for the P42/ncm phase. The refinement also shows that the crystals are stoichiometric with an O compn. of 4.02 .+-. 0.03. The crystals are antiferromagnetic with a magnetic structure in which the propagation and spin directions are parallel to [100], in agreement with other studies. The antiferromagnetic ordered moment at 10 K is (1.62 .+-. 0.05) .mu.B per Ni atom. No change in the antiferromagnetic structure occurs at the 70-K transition, but one can not exclude the formation of a 2k magnetic structure at this temp. that would give rise to the same diffraction pattern in the tetragonal phase. The induced magnetic form ***factor*** (***H*** -4.6 T, applied parallel to the [010] axis at 5 K) was measured with polarized neutrons. There was measured 18 reflections with 3 different neutron wavelengths. The induced moment is 7.9 m.mu.B per mol., which is in good agreement with bulk susceptibility measurements. There is no evidence for any spin transfer or asphericity in the Ni spin d. in the Ni-O planes. Instead one obsd. new effects along the long c axis. The La3+ atoms develop a pos. susceptibility which may come from ***hybridization*** between the La 5d and O p bands.

L32 ANSWER 29 OF 51 MEDLINE DUPLICATE 9
AN 90049900 MEDLINE
TI Monoclonal antibodies directed to the calcium-free conformation of human protein S.
AU Marcovina S; Coppola R; Valsecchi C; Zoppo A; Gelfi C; Mannucci P M
CS Scientific Institute Hospital S. Raffaele, Milan, Italy..
SO THROMBOSIS AND HAEMOSTASIS, (1989 Sep 29) 62 (2) 708-14.
Journal code: VQ7. ISSN: 0340-6245.
CY GERMANY, WEST: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9002
AB Four mouse ***hybridomas*** secreting monoclonal antibodies specific for human protein S (PS) have been generated. The antibodies, all of the IgG1 subclass, were designated S2, S3, S8, and S10. In a fluid phase radioimmunoassay, the binding of monoclonal antibodies to PS was about 30% greater in the presence of EDTA and totally inhibited in presence of Ca2+. Using the same technique, we performed displacement curves of 125I-labeled PS by purified PS, thrombin-cleaved PS, normal plasma, plasma from a patient on warfarin therapy, and plasma from a patient with no free PS and only PS bound to ***C4b*** - ***binding*** ***protein*** . The slopes of the curves show that the monoclonal antibodies reacted equally with all the tested forms of PS indicating that the antigenic site(s) to which the monoclonal antibodies are directed are present and exposed in free and bound PS, in thrombin-cleaved PS, and in the coumarin form of the protein. Each EDTA-dependent antibody, immobilized on Sepharose 4B-CNBr was used to purify PS from the barium citrate-absorbed, ammonium

sulphate-soluble fraction of plasma. The fraction eluted from the immunoabsorbent with a buffer containing 4 mmol/l CaCl2 and analysed by SDS-PAGE, contained two bands, one migrating with conventionally purified PS and the other with purified ***C4b*** - ***binding*** ***protein*** . Homogeneous PS was obtained by chromatography of the barium citrate adsorbate on a DEAE-Sephadex column. The protein peak containing the bulk of PS was subsequently applied to the immunoadsorbent and eluted with 4 mmol/l CaCl2.(ABSTRACT TRUNCATED AT 250 WORDS)

L32 ANSWER 30 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1989:93240 HCAPLUS
DN 110:93240
TI Identification of murine complement receptor type 2
AU Fingeroth, Joyce D.; Benedict, Mary A.; Levy, David N.; Strominger, Jack L.
CS Div. Infect. Dis., Dana-Farber Cancer Inst., Boston, MA, 02115, USA
SO Proc. Natl. Acad. Sci. U. S. A. (1989), 86(1), 242-6
CODEN: PNASA6; ISSN: 0027-8424
DT Journal
LA English
AB A rabbit antiserum reactive with the human complement component C3d/Epstein-Barr virus receptor (complement receptor type 2, CR2) immunoppts. a Mr 155,000 murine B-cell surface antigen. The apparent mol. wt. and cellular distribution of this murine antigen are similar to those of human CR2. Cells expressing the murine protein bind sheep erythrocytes coated with antibody and murine C1-C3d but do not bind Epstein-Barr virus. The monospecific antiserum to human CR2 together with goat F(ab')2 anti-rabbit IgG blocks attachment of the C3d-coated erythrocytes to receptor-bearing murine B lymphocytes. To further characterize murine CR2, a .lambda.gt11 library from the murine late pre-B-cell line 70Z/3 was screened with human CR2 cDNA. A partial cDNA clone of 3.5 kilobases with 79% amino acid sequence identity to human CR2 in the unique intracytoplasmic region and 63% identity to the sixth human CR2 repeat was obtained. Blot ***hybridization*** with the murine cDNA clone identified an RNA species of .apprxeq.4.7 kilobases, similar in size to human CR2 mRNA from a murine B-cell line but not from a murine T-cell line.

L32 ANSWER 31 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1990:16950 HCAPLUS
DN 112:16950
TI Characterization of the structural genes for the DNA-binding protein H-NS in Enterobacteriaceae
AU La Teana, A.; Falconi, M.; Scarlato, V.; Lammi, M.; Pon, C. L.
CS Lab. Genet., Univ. Camerino, Camerino, 62032, Italy
SO FEBS Lett. (1989), 244(1), 34-8
CODEN: FEBLAL; ISSN: 0014-5793
DT Journal
LA English
AB The promoter region of Escherichia coli hns, the structural gene for the DNA-binding protein H-NS, was identified by use of a promoter search vector and the in vivo transcriptional start point was detd. by primer extension anal. The homologous hns genes of 2 other Enterobacteriaceae, Proteus vulgaris and Serratia marcescens, were identified by heterologous ***hybridization*** with a DNA probe derived from E. coli hns, cloned and sequenced. Taking into account only the invariant nucleotides and amino acids, the homol. of H-NS among the 3 organisms was found to be >70% at the DNA level and >75% at the protein level. The 3 hns genes were also found to have nearly identical transcriptional and translational signals.

L32 ANSWER 32 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1989:422033 HCAPLUS
DN 111:22033
TI Immunological determination of free human protein S and C4bp-protein S complex using monoclonal antibodies, and an immunosorbent for separation or recovery of protein S
IN Koike, Yukiya; Wakabayashi, Kenji; Sumi, Yoshihiko; Ichikawa, Yataro
PA Teijin Ltd., Japan
SO Eur. Pat. Appl., 19 pp.
CODEN: EPXXDW
PI EP 271810 A2 880622
DS R: BE, CH, DE, FR, GB, LI, SE
AI EP 87-118183 871208
PRAI JP 86-296766 861215
JP 86-298881 861217
DT Patent
LA English
AB Two solid-phase sandwich immunoassay methods are described for detn. of free human protein S and of a complex of human protein S and human complement ***C4b*** - ***binding*** ***protein*** (C4bp), resp. Free protein S is detd. with a primary immobilized antibody and a labeled secondary antibody which bind to different epitopes of protein S; 1 of these is a monoclonal antibody (MAb) which specifically binds to free protein S but not to the complex. In detn. of the complex, 1 of the antibodies is a MAb and binds specifically to the complex but not to free protein S or C4bp, and the other binds to C4bp. An immobilized MAb may be used as selective adsorbent for sepn. or recovery of protein S from a liq. Mice were immunized with purified protein S, and their spleen cells were used to prep. ***hybridomas*** for prodn. of MAbs. Clones were isolated which secreted MAbs (e.g. 2B9F12) with high specificity for free protein S; 1 MAb (2E12C7) bound very weakly to free protein S, but showed high specificity for the C4bp-protein S complex. MAb 2B9F12 was coated on a microtiter plate which was then exposed to purified protein S and plasma sample. The plate was washed and incubated with peroxidase-labeled MAb 2B9C10 (specific for protein S) followed by substrate (ABTS), and the change of absorbance was measured at 415 nm for detn. of protein S.

L32 ANSWER 33 OF 51 MEDLINE DUPLICATE 10
AN 88217940 MEDLINE
TI Glycolipid reanchoring of T-lymphocyte surface antigen CD8 using the 3' end sequence of ***decay*** - ***accelerating*** ***factor*** 's mRNA.
AU Tykocinski M L; Shu H K; Ayers D J; Walter E I; Getty R R; Groger R K; Hauer C A; Medof M E
CS Institute of Pathology, Case Western Reserve University, Cleveland, OH 44106..
NC AI23598
AI24220
CA43703
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES
OF AMERICA, (1988 May) 85 (10) 3555-9.
Journal code: PV3. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 8808
AB ***Decay*** - ***accelerating*** ***factor*** (DAF) is one of a family of cell-associated proteins that undergo posttranslational modifications in which glycolipid anchoring structures are substituted for membrane-spanning sequences. The signals that direct the covalent substitution reaction in these proteins are unknown. Human DAF was expressed in Chinese hamster ovary (CHO) cells and murine BW lymphocytes. In both cases, the xenogeneic DAF in transfectants incorporated a glycolipid anchor. A ***chimeric*** CD8-DAF cDNA, encompassing the extra-cellular region of the T-lymphocyte surface antigen CD8 and the 3' end of DAF mRNA (encoding the C-terminal region of mature DAF as well as the hydrophobic extension peptide), was expressed in human leukemia lines after transfection with an Epstein-Barr virus-based episomal vector. The ***chimeric*** protein in transfectants demonstrated glycolipid anchoring, whereas unaltered CD8 in control experiments did not. The signals directing glycolipid anchoring in eukaryotic cells are thus evolutionarily conserved and contained in the 3' end of the DAF sequence.

L32 ANSWER 34 OF 51 MEDLINE DUPLICATE 11
AN 88124944 MEDLINE
TI Normal polymorphic variations and transcription of the ***decay*** ***accelerating*** ***factor*** gene in paroxysmal nocturnal hemoglobinuria cells.
AU Stafford H A; Tykocinski M L; Lublin D M; Holers V M; Rosse W F; Atkinson J P; Medof M E
CS Institute of Pathology, Case Western Reserve University, Cleveland, OH 44106..
NC AI23598
AI24220
AI19642
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES
OF AMERICA, (1988 Feb) 85 (3) 880-4.
Journal code: PV3. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 8805
AB In paroxysmal nocturnal hemoglobinuria (PNH), an acquired hemolytic anemia, deficiency of ***decay*** ***accelerating*** ***factor*** (DAF) renders blood cells susceptible to increased deposition of autologous complement activation fragments (C3b) and complemented-mediated injury. To investigate the mechanism of the DAF defect, DNA and mRNA from normal and PNH leukocytes were compared in blot ***hybridization*** assays by using DAF cDNA and oligonucleotide probes. Southern analyses of DNA from normal cells revealed a single gene spanning approximately equal to 35 kilobases of DNA. Six HindIII banding patterns were distinguishable among normal individuals. In family studies, the patterns segregated as three homozygous and three heterozygous genotypes deriving from three haplotypes: A, B, and C with frequencies of 0.47, 0.36, and 0.17, respectively. Oligonucleotide mapping localized the polymorphic HindIII sites to two noncoding regions in the vicinity of exons encoding (i) the protein oligosaccharide-rich domain and (ii) the mRNA 3'-untranslated region. Analyses of DNA from DAF-negative leukocytes of eight PNH patients demonstrated restriction fragment profiles identical to those of normal individuals for all enzymes studied. Three patients had the BC (normals = 3/32), three patients had the AA (normals = 6/32), and two patients had the AC (normals = 8/32) HindIII genotype. Of the three PNH patients exhibiting the BC genotype, family studies of two

demonstrated the expected inheritance patterns, and RNA gel blot analyses of two showed mRNA transcripts indistinguishable from those in normal cells. The absence of DAF gene or mRNA alterations in affected PNH cells that lack other glycolipid-anchored proteins as well as DAF argues that the lesion underlying PNH cells resides in the glycolipid-anchor pathway.

L32 ANSWER 35 OF 51 MEDLINE DUPLICATE 12
AN 88242821 MEDLINE
TI Derivation of the sequence of the signal peptide in human ***C4b*** - ***binding*** ***protein*** and interspecies cross- ***hybridisation*** of the C4bp cDNA sequence.
AU Lintin S J; Lewin A R; Reid K B
CS Department of Biochemistry, University of Oxford, England..
SO FEBS LETTERS, (1988 May 23) 232 (2) 328-32.
Journal code: EUH. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK-X07853
EM 8809
AB A 5' cDNA clone coding for human ***C4b*** - ***binding*** ***protein*** (C4bp) was isolated, characterised and sequenced to complete the cDNA sequence coding for residues 1-32 thus confirming the protein sequence data of Chung et al. [(1985) Biochem. J. 230, 133-141]. The sequence extended to allow derivation of the putative leader peptide sequence which was 32 residues in length and showed a high of hydrophobicity typical of other documented leader sequences. Cross ***hybridisation*** was detected between the human C4bp cDNA probes and genomic DNA isolated from various species on Southern blots suggesting that genomic sequence homologous to that coding for C4bp has been conserved during evolution.

L32 ANSWER 36 OF 51 MEDLINE DUPLICATE 13
AN 88286080 MEDLINE
TI Molecular cloning and chromosomal localization of human ***membrane*** ***cofactor*** ***protein*** (MCP). Evidence for inclusion in the multigene family of complement-regulatory proteins.
AU Lublin D M; Liszewski M K; Post T W; Arce M A; Le Beau M M; Rebentisch M B; Lemons L S; Seya T; Atkinson J P
CS Department of Pathology, Washington University, St. Louis, Missouri 63110..
NC AI-19642
AI-15322
GM-35377
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1988 Jul 1) 168 (1) 181-94.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK-Y00651
EM 8811
AB ***Membrane*** ***cofactor*** ***protein*** (MCP), a regulatory molecular of the complement system with cofactor activity for the factor I-mediated inactivation of C3b and C4b, is widely distributed, being present on leukocytes, platelets, endothelial cells, epithelial cells, and fibroblasts. MCP was purified from a human T cell line (HSB2) and the NH2-terminal 24-amino acid sequence obtained by Edman degradation. An oligonucleotide probe based on this sequence was used to identify a clone from a human monocytic (U937) cDNA library. Nucleotide sequencing showed a 43-bp 5'-untranslated region, an open reading frame of 1,152 bp, and a 335-bp 3'-untranslated region followed by a 16-bp poly(A) track. The deduced full-length MCP protein consists of a 34-amino acid signal peptide and a 350-amino acid mature protein. The protein has, beginning at the NH2 terminus, four approximately 60-amino acid repeat units that match the consensus sequence found in a multigene family of complement regulatory proteins (C3b-receptor or CR1, C3d-receptor or CR2, ***decay*** - ***accelerating*** ***factor*** , ***C4*** - ***binding*** ***protein*** , and ***factor*** ***H***), as well as several other complement and non-complement proteins. The remainder of the MCP protein consists of 25 amino acids that are rich in serine and threonine (probable site of heavy O-linked glycosylation of MCP), 17 amino acids of unknown significance, and a 23-amino acid transmembrane hydrophobic region followed by a 33-amino acid cytoplasmic tail. The MCP gene was localized to human chromosome 1, bands 1q31-41, by analysis of human x rodent somatic cell ***hybrid*** clones and by in situ ***hybridization*** . This same genetic region contains the multigene family of complement-regulatory proteins, which is thereby enlarged to include the functionally and structurally related MCP.

L32 ANSWER 37 OF 51 MEDLINE DUPLICATE 14
AN 89245795 MEDLINE
TI Assignment of complement components ***C4*** ***binding*** ***protein*** (C4BP) and ***factor*** ***H*** (FH) to human chromosome 1q, using cDNA probes.
AU Hing S; Day A J; Linton S J; Ripoche J; Sim R B; Reid K B; Solomon E
CS Imperial Cancer Research Fund, Lincoln's Inn Fields, London..
SO ANNALS OF HUMAN GENETICS, (1988 May) 52 (Pt 2) 117-22.
Journal code: 58C. ISSN: 0003-4800.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 8908
AB Using cDNA probes for ***Factor*** ***H*** (FH) and ***C4*** ***binding*** ***protein*** (C4BP) on a panel of somatic cell ***hybrids*** , we show that both of these genes map to the long arm of chromosome 1.

L32 ANSWER 38 OF 51 MEDLINE
AN 88024997 MEDLINE
TI cDNA structure of murine ***C4b*** - ***binding*** ***protein*** , a regulatory component of the serum complement system.
AU Kristensen T; Ogata R T; Chung L P; Reid K B; Tack B F
CS Department of Immunology, Scripps Clinic and Research Foundation, La Jolla, California 92037..
NC AI 19222
AI 22214
GM 29831
SO BIOCHEMISTRY, (1987 Jul 28) 26 (15) 4668-74.
Journal code: A0G. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-M17122
EM 8802
AB A cDNA library representing total poly(A+) RNA from the livers of male B10.WR mice was screened with a 1097 base pair (bp) probe obtained from a partial human ***C4b*** - ***binding*** ***protein*** (C4BP) cDNA clone. Two cDNA clones were isolated, the largest of which was sequenced and found to be 1889 bp in length exclusive of the poly(A) tail. The predicted mouse C4BP polypeptide chain encoded by 1239 bp is 413 amino acid residues in length and has a calculated molecular weight of 45,281. The 370-nucleotide sequence upstream from the codon for the predicted amino terminus contains two possible in-phase translational start signals which yield leader sequences of 56 and 13 amino acid residues, respectively. The 3'-untranslated region is 277 bp long, and there are two potential overlapping poly(A) recognition signals, AATTAA and ATTAAAA, located 26 and 25 bp, respectively, upstream from the poly(A) tail; these are preceded by five other potential polyadenylation signals. Beginning at the amino terminus and continuing through to residue 358, there are six contiguous regions of internal homology, each about 60 amino acids in length. The carboxy-terminal 55 amino acid sequence shares no homology with the repeating units. Extensive homology was found with human C4BP at the amino acid level (61%) as well as at the nucleotide level for both the coding and 3'-untranslated regions. Significant differences, however, were observed between mouse and human C4BP.(ABSTRACT TRUNCATED AT 250 WORDS)

L32 ANSWER 39 OF 51 MEDLINE DUPLICATE 15
AN 87196375 MEDLINE
TI Expansion of the complement receptor gene family. Identification in the mouse of two new genes related to the CR1 and CR2 gene family.
AU Aegerter-Shaw M; Cole J L; Klickstein L B; Wong W W; Fearon D T; Lalley P A; Weis J H
NC AI-23401
AI-22833
AI 07323-01
+
SO JOURNAL OF IMMUNOLOGY, (1987 May 15) 138 (10) 3488-94.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
OS GENBANK-M16179
EM 8708
AB Human cDNA probes encoding the C3b/C4b complement receptor, CR1, have been used to identify, in the mouse, two new genes which are related to CR1 but which appear to encode a different protein product. These new mouse genes, arbitrarily designated mouse genes X and Y, ***hybridize*** specifically to three different cDNA probes derived from human CR1. The degree of ***hybridization*** homology between the mouse X and Y genes suggests they are very closely related to one another; however, the chromosomal localization of the mouse X gene to chromosome 8 and the mouse Y gene to chromosome 1 indicates they are distinct gene sequences. The mRNA species detected with the X and/or Y (X/Y) sequences are approximately 2000 bases in length, but vary in both quantity and size depending upon the tissue analyzed. DNA sequence analysis of a cDNA specific for the X and Y sequences indicates the mature protein(s) will contain the 60 amino acid consensus repeat characteristic of a group of other proteins including CR1, the C3d receptor (CR2), H, ***C4*** ***binding*** ***protein*** (C4bp), the interleukin 2 (Il 2) receptor and others. The identity of the mouse X and Y genes, and the function of the proteins which

they encode, is not known; however, the small size of the mRNA and the tissue specific expression suggests they do not encode mouse CR1 or CR2 but instead encode a related protein (or proteins) which is expressed in a wide variety of mouse tissues.

L32 ANSWER 40 OF 51 MEDLINE DUPLICATE 16
AN 87224744 MEDLINE
TI The gene encoding ***decay*** - ***accelerating*** ***factor*** (DAF) is located in the complement-regulatory locus on the long arm of chromosome 1.
AU Lublin D M; Lemons R S; Le Beau M M; Holers V M; Tykocinski M L; Medof M E; Atkinson J P
NC AI19642
AI15322
GM35377
+
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1987 Jun 1) 165 (6) 1731-6.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 8709
AB Delay-accelerating factor (DAF) protects host cells from complement-mediated damage by regulating the activation of C3 convertases on host cell surfaces. Using a panel of hamster-human somatic cell ***hybrids*** , the DAF gene was mapped to human chromosome 1. In situ ***hybridization*** studies using human metaphase cells further localized the gene to bands 1q31-41, with the largest cluster of grains at 1q32. This establishes the close linkage of the DAF gene to genes for four other proteins (C3b/C4b receptor or ***complement*** ***receptor*** ***1*** , C3d receptor or ***complement*** ***receptor*** ***2*** , ***factor*** ***H*** , and ***C4*** - ***binding*** ***protein***) that share 60-amino-acid homologous repeats as well as complement-regulatory or -receptor activity, thereby enlarging the complement-regulatory gene family on the long arm of human chromosome 1.

L32 ANSWER 41 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1987:405439 HCAPLUS
DN 107:5439
TI Separation of the genetic loci for the H-Y antigen and for testis determination on human Y chromosome
AU Simpson, Elizabeth; Chandler, Phillip; Goulmy, Els; Disteche, Christine M.; Ferguson-Smith, Malcolm A.; Page, David C.
CS Transplant. Biol. Sect., Clin. Res. Cent., Harrow/Middlesex, HA1 3UJ, UK
SO Nature (London) (1987), 326(6116), 876-8
CODEN: NATUAS; ISSN: 0028-0836
DT Journal
LA English
AB The mammalian Y chromosome encodes a testis-detg. factor (termed TDF in the human), a master regulator of sex differentiation. Expression of H-Y, a minor histocompatibility antigen, may also be controlled by a gene on the Y chromosome, and it has been proposed that this antigen is the testis-detg. factor. The authors tested the postulated identity of H-Y and TDF in the human. H-Y typing with T cells was carried out on a series of sex-reversed humans (XX males and XY females), each shown by DNA ***hybridization*** to carry part but not all of the Y chromosome. This deletion anal. maps the gene for H-Y to the long arm or centromeric region of the human Y chromosome, far from the TDF locus, which maps to the distal short arm. Thus, the H-Y antigen is not the testis-detg. factor.

L32 ANSWER 42 OF 51 MEDLINE
AN 87059049 MEDLINE
TI A complement receptor locus: genes encoding C3b/C4b receptor and C3d/Epstein-Barr virus receptor map to 1q32.
AU Weis J H; Morton C C; Bruns G A; Weis J J; Klickstein L B; Wong W W; Fearon D T
NC AI-23401
AI-22833
HO18658
+
SO JOURNAL OF IMMUNOLOGY, (1987 Jan 1) 138 (1) 312-5.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 8703
AB The alternative or classical pathways for complement system component C3 may be triggered by microorganisms and antigen-antibody complexes. In particular, an activated fragment of C3, C3b, covalently attaches to microorganisms or antigen-antibody complexes, which in turn bind to the C3b receptor, also known as ***complement*** ***receptor*** ***1*** . The genes encoding the proteins that constitute the C3-activating enzymes have been cloned and mapped to a "complement activation" locus in the major histocompatibility complex, and we demonstrate in this study such a locus on the long arm of chromosome 1 at band 1q32.

L32 ANSWER 43 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1987:475949 HCAPLUS
DN 107:75949
TI Monoclonal antibody to ***decay*** ***accelerating*** ***factor*** (DAF), a method for making it, and use
IN Kinoshita, Taroh; Medof, M. Edward; Nussenzweig, Victor
PA New York University, USA
SO PCT Int. Appl., 45 pp.
CODEN: PIXXD2
PI WO 8607062 A1 861204
DS W: AU, JP
RW: AT, BE, CH, DE, FR, GB, IT, LU, NL, SE
AI WO 86-US1177 860523
PRAI US 85-738171 850524
DT Patent
LA English
AB DAF, and erythrocyte membrane protein which may protect erythrocytes from damage by autologous complement, is purified to homogeneity by a combination of anion-exchange and hydrophobic interaction chromatog., gel filtration, lectin-Sepharose chromatog., immunoaffinity chromatog., and HPLC. Purified DAF is used to induce formation of a monoclonal antibody by the ***hybridoma*** method. The anti-DAF antibody enhances the complement-induced lysis of cells (e.g. tumor cells) previously exposed to an antibody to a cell surface antigen. It is also useful for immunoassays for DAF and for identification of DAF-pos. cells, e.g. with a fluorescence-activated cell sorter and fluorescein-conjugated anti-Ig. Patients with paroxysmal nocturnal hemoglobinuria showed an increasing fraction of DAF-neg. erythrocytes with increasing severity of the disease; leukocytes and platelets were also deficient in DAF in these patients.

L32 ANSWER 44 OF 51 MEDLINE DUPLICATE 17
AN 86287311 MEDLINE
TI Identification of a partial cDNA clone for the C3d/Epstein-Barr virus receptor of human B lymphocytes: homology with the receptor for fragments C3b and C4b of the third and fourth components of complement.
AU Weis J J; Fearon D T; Klickstein L B; Wong W W; Richards S A; de Bruyn Kops A; Smith J A; Weis J H
NC GM07753-06
AI23401
AI22833
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES
OF AMERICA, (1986 Aug) 83 (15) 5639-43.
Journal code: PV3. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 8611
AB Human complement receptor type 2 (CR2) is the B-lymphocyte receptor both for the C3d fragment of the third component of complement and for the Epstein-Barr virus. Amino acid sequence analysis of tryptic peptides of CR2 revealed a strong degree of homology with the human C3b/C4b receptor, CR1. This homology suggested that CR1 gene sequences could be used to detect the CR2 sequences at conditions of low-stringency ***hybridization*** . Upon screening a human tonsillar cDNA library with CR1 cDNA sequences, two clones were identified that ***hybridized*** at low, but not at high, stringency. Redundant oligonucleotides specific for CR2 sequences were synthesized and used to establish that the two cDNA clones weakly ***hybridizing*** with the CR1 cDNA contained CR2 sequences. One of these CR2 cDNA clones ***hybridized*** to oligonucleotides derived from two distinct CR2 tryptic peptides, whereas the other, smaller cDNA clone ***hybridized*** to oligonucleotides derived from only one of the CR2 peptides. Nucleotide sequence analysis of the CR2 cDNA confirmed that the site of oligonucleotide ***hybridization*** was identical to that predicted from the peptide sequence, including flanking sequences not included within the oligonucleotide probes. The CR2-specific cDNA sequences identified a poly(A)+ RNA species of 5 kilobases in RNA extracted from human B cells but did not ***hybridize*** to any RNA obtained from the CR2-negative T-cell line HSB-2, thus confirming the appropriate size and tissue-specific distribution for the CR2 mRNA. The striking peptide sequence homology between CR2 and CR1 and the cross- ***hybridization*** of the CR2 cDNA with the CR1-specific sequences allow the placement of CR2 in a recently defined gene family of C3- and C4-binding proteins consisting of CR1, ***C4*** - ***binding*** ***protein*** , ***factor*** ***H*** , and now, CR2.

L32 ANSWER 45 OF 51 MEDLINE DUPLICATE 18
AN 87059010 MEDLINE
TI Chromosomal location of the genes encoding complement components C5 and ***factor*** ***H*** in the mouse.
AU D'Eustachio P; Kristensen T; Wetsel R A; Riblet R; Taylor B A; Tack B F
NC GM32105
AI17354
AI22214
+

SO JOURNAL OF IMMUNOLOGY, (1986 Dec 15) 137 (12) 3990-5.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 8703
AB Complementary DNA probes corresponding to the ***factor*** ***H*** and C5 polypeptides have been used to determine the chromosomal localizations of these two complement components. Both probes revealed complex and polymorphic arrays of DNA fragments in Southern blot analysis of mouse genomic DNA. Following the distribution of these bands in panels of somatic cell ***hybrids*** carrying various combinations of mouse chromosomes on a constant rat or Chinese hamster background allowed the localization of the C5-associated fragments to proximal chromosome 2 and the localization of the ***factor*** ***H*** -associated fragments to chromosome 1 or chromosome 3. Following the inheritance of DNA restriction fragment-length polymorphisms revealed by the probes in recombinant inbred mouse strains allowed the ***factor*** ***H*** -associated fragments to be mapped to Sas-1 on chromosome 1, and the C5-associated fragments to be mapped to Hc. Analysis of three-point crosses, in turn, placed the latter locus 19 cM distal to Sd on chromosome 2. We have designated the two loci Cfh and C5, respectively. This genetic analysis raises the possibility that C5 and ***factor*** ***H*** are both encoded by complex loci composed of distinct structural and regulatory genes.

L32 ANSWER 46 OF 51 MEDLINE DUPLICATE 19
AN 86233353 MEDLINE
TI Murine protein H is comprised of 20 repeating units, 61 amino acids in length.
AU Kristensen T; Tack B F
NC AI 19222
AI 22214
AI 17354
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES
OF AMERICA, (1986 Jun) 83 (11) 3963-7.
Journal code: PV3. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK-M12660
EM 8609
AB A cDNA library constructed from size-selected (greater than 28 S) poly(A)+ RNA isolated from the livers of C57B10. WR mice was screened by using a 249-base-pair (bp) cDNA fragment encoding 83 amino acid residues of human protein H as a probe. Of 120,000 transformants screened, 30 ***hybridized*** with this cDNA probe. Ten positives were colony-purified, and the largest plasmid cDNA insert, MH8 (4.4 kb), was sequenced by the dideoxy chain termination method. MH8 contained the complete coding sequence for the precursor of murine complement protein ***factor*** ***H*** (3702 bp), 100 bp of 5'-untranslated sequence, 448 bp of 3'-untranslated sequence, and a polyadenylylated tail of undetermined length. Murine pre-protein H was deduced to consist of an 18-amino acid signal peptide and 1216 residues of H-protein sequence. Murine H was composed of 20 repetitive units, each about 61 amino acid residues in length. Similar repetitive units are present in the ***C4b*** ***binding*** ***protein*** , the C3b-receptor (CR1), complement factor B and C2, and in beta 2-glycoprotein I and the interleukin 2 receptor. This finding suggests a common evolutionary origin for regions of these proteins.

L32 ANSWER 47 OF 51 MEDLINE DUPLICATE 20
AN 86169701 MEDLINE
TI Structural analysis of human complement protein H: homology with ***C4b*** ***binding*** ***protein*** , beta 2-glycoprotein I, and the Ba fragment of B2.
AU Kristensen T; Wetsel R A; Tack B F
NC AI 19222
AI 22214
SO JOURNAL OF IMMUNOLOGY, (1986 May 1) 136 (9) 3407-11.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
OS GENBANK-M12383
EM 8607
AB We report here a partial primary structure for human complement protein H. Tryptic peptides comprising 27% of the H molecule were isolated by conventional techniques and were sequenced (333 amino acid residues). Several mixed-sequence oligonucleotide probes were constructed, based on the peptide sequence data, and were used to screen a human liver cDNA library. The largest recombinant plasmid (pH1050), which ***hybridized*** with two probes, was further characterized. The cDNA insert of this plasmid contained coding sequence (672 bp) for 224 amino acids of H. The 3' end of this clone had a polyadenylated tail preceded by a polyadenylation recognition site (ATTAAA) and a 3'-untranslated region (229 bp). Four regions of internal homology, each about 60 amino acids in length, were observed in the derived protein sequence from this cDNA clone, and a further seven from the tryptic peptide sequences. The consensus sequence for each of the repetitive units of H was four cysteines, two prolines, three glycines, one tryptophan, and two tyrosines/phenylalanines. Based on the mole percent values for each of these amino acids, it is likely that H is composed of about 20 repetitive units of this nature. Furthermore, the repetitive unit of H shows pronounced homology with the Ba fragment of B, the ***C4b*** ***binding*** ***protein*** , and beta 2-glycoprotein I. Therefore, it seems that at least portions of these proteins have evolved from a common ancestral DNA element.

L32 ANSWER 48 OF 51 MEDLINE DUPLICATE 21
AN 86216621 MEDLINE
TI Monoclonal antibodies to human vitamin K-dependent protein S.
AU Litwiller R D; Jenny R J; Katzmann J A; Miller R S; Mann K G
NC HL 17430
HL16150
SO BLOOD, (1986 Jun) 67 (6) 1583-90.
Journal code: A8G. ISSN: 0006-4971.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 8609
AB Monoclonal antibodies to human protein S have been prepared using established ***hybridoma*** technology. One antibody was isolated that binds protein S only when Ca2+ is present; others bind antigen equally well in the presence or absence of EDTA. Other antibodies display a diminished affinity for protein S in the presence of EDTA. Purified immunoglobulins from cell lines displaying Ca2+ dependence were immobilized and used to purify protein S from fractions obtained by barium precipitation of citrated plasma, ammonium sulfate fractionation, and chromatography on diethylaminoethanol (DEAE)-Sephadex and dextran sulfate agarose. Essentially homogeneous protein S was isolated from the barium-citrate-adsorbed, 35% ammonium-sulfate-soluble proteins using a totally Ca2+-dependent antibody and EDTA elution. Protein S and several substances of higher mol wt were bound directly from plasma by a partially Ca2+-dependent antibody and were eluted partially with EDTA and NaCl and finally with NaSCN. The largest and most abundant of the high mol wt materials is likely protein S-complement ***C4b*** - ***binding*** ***protein*** complex. The immunoaffinity-isolated protein S was found to be indistinguishable from conventionally isolated protein S in terms of activity, sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) mobility, and by high-performance liquid chromatography (HPLC). These studies establish reagents that can probe the structure of protein S and isolate protein S in its free and complexed forms.

L32 ANSWER 49 OF 51 HCAPLUS COPYRIGHT 1997 ACS
AN 1987:28379 HCAPLUS
DN 106:28379
TI Analysis of multiple restriction fragment length polymorphisms of the gene for the human complement receptor type I. Duplication of genomic sequences occurs in association with a high molecular mass receptor allotype
AU Wong, Winnie W.; Kennedy, Christine A.; Bonaccio, Ermelinda T.; Wilson, James G.; Klickstein, Lloyd B.; Weis, John H.; Fearon, Douglas T.
CS Dep. Rheumatol. Immunol., Brigham and Women's Hosp., Boston, MA, 02115, USA
SO J. Exp. Med. (1986), 164(5), 1531-46
CODEN: JEMEAV; ISSN: 0022-1007
DT Journal
LA English
AB Human complement receptor type 1 (CR1) exhibits an unusual form of polymorphism in which allotypic variants differ in the mol. wt. of their resp. polypeptide chains. To address mechanisms involved in the generation of the CR1 allotypes, DNA from individuals having the F allotype (250,000 Mr), the S allotype (290,000 Mr), and the F' allotype (210,000 Mr) was digested by restriction enzymes, and Southern blots were ***hybridized*** with CR1 cDNA and genomic probes. With the use of BamHI and SacI, an addnl. restriction fragment was obsd. in 20 of 21 individuals having the S allotype with no assocd. loss of other restriction fragments. Southern blot anal. with a noncoding genomic probe derived from the S allotype-specific BamHI fragment showed ***hybridization*** to this fragment and to 2 other fragments that were also present in FF individuals. Thus, an intervening sequence may be repeated twice in the F allele and 3 times in the S allele. A restriction fragment length polymorphism (RFLP) unique to 2 individuals expressing the F' allotype was seen with EcoRV, but the absence of persons homozygous for this rare allotype prevented further comparisons with the F and S allotypes. Anal. of the CR1 transcripts assocd. with the 3 CR1 allotypes indicated that these differed by 1.3-1.5 kb and had the same rank order as the corresponding allotypes. Taken together, these findings suggest that the S allele was generated from the F allele by the acquisition of addnl. sequences, the coding portion of which may correspond to a long homologous repeat of .apprx.1.4 kb that has been identified in CR1 cDNA. Two other RFLPs with HindIII

and PuvII that were in linkage disequil. with the BamHI-SacI RFLPs assocd. with the S allotype, and a 3rd polymorphism was seen with EcoRI that was not in linkage disequil. with the other polymorphisms were obsd. Thus, 10 commonly occurring CR1 alleles can be defined, making this locus a useful marker for the long arm of chromosome 1 to which the CR1 gene maps.

L32 ANSWER 50 OF 51 MEDLINE DUPLICATE 22
AN 85132706 MEDLINE
TI Structural polymorphism of murine complement ***factor*** ***H*** controlled by a locus located between the Hc and the beta 2M locus on the second chromosome of the mouse.
AU Natsuume-Sakai S; Sudoh K; Kaidoh T; Hayakawa J; Takahashi M
SO JOURNAL OF IMMUNOLOGY, (1985 Apr) 134 (4) 2600-6.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 8506
AB Structural polymorphism of murine ***factor*** ***H*** protein was demonstrated by using three different methods. 1) By prolonged agarose electrophoresis and immunofixation, ***factor*** ***H*** protein was visualized in the beta region as a single, distinct protein band in freshly bled EDTA-plasmas from many laboratory and wild mice. Two variants were detected among a large number of tested strains; one, referred to as H.1, moved faster to the anodal region (type strain, BALB/c), and the other, referred to as H.2, moved more slowly to the anodal region (type strain, STR). The F1 ***hybrid*** between BALB/c and STR exhibited a combining type of ***factor*** ***H*** protein, which was observed in each parent. 2) Two-dimensional peptide mapping analysis was carried out with tryptic peptides of these two ***factor*** ***H*** allotypes. Almost all of the spots in the maps of tryptic peptides were common to both allotypes. However, three distinct spots among the 57 spots detected in the map of tryptic peptides of the H.1 allotypes were not detected in that of H.2 allotype, whereas two spots among the 56 spots in the map of H.2 allotype were unique for this allotype. The F1 ***hybrid*** between BALB/c and STR showed a combining type of the map of parent. 3) Alloantisera against each of H allotypes were successfully produced in BALB/c or BALB/c-H.2 (a congenic strain with H.2 allotype) by repeated injection of each purified ***factor*** ***H*** protein either from the BALB/c or the STR strain. These findings indicated that the observed variants of ***factor*** ***H*** represent antigenically and structurally distinguishable allotypes. The allotypes of murine ***factor*** ***H*** protein are controlled by a single codominant locus located between the Hc locus and the beta 2M locus on the second chromosome of the mouse. This was shown by phenotyping the Hc locus and H locus with backcross progenies between A/J (one of strain with H.1) and MoA (one of strain with H.2). The recombination frequency between these two loci was 0.17 +/- 0.046.

L32 ANSWER 51 OF 51 MEDLINE
AN 84287603 MEDLINE
TI Monoclonal antibody to human epidermal growth factor.
AU Moriarity D M; Harper R A; Knowles B B; Savage C R Jr
NC GM 29257
AM 25436
CA 18470
+
SO HYBRIDOMA, (1983) 2 (3) 321-7.
Journal code: GFS. ISSN: 0272-457X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 8412
AB A monoclonal antibody directed to a species-specific determinant of human epidermal growth ***factor*** (***h*** -EGF) was obtained by fusing murine myeloma cells with BALB/c mouse splenocytes sensitized to h-EGF. This antibody, referred to as 863.D4, did not react with either rat or mouse epidermal growth factor or with 11 other polypeptide hormones tested as shown by solid-phase radioimmunoassay (SPRIA), and immunoprecipitation followed by sodium dodecylsulfate polyacrylamide gel electrophoresis (SDS-PAGE). Scatchard analysis of the antibody binding to purified h-EGF revealed an apparent equilibrium dissociation constant of 1 X 10(-8) M. The antibody blocked both the binding of h-EGF and h-EGF stimulation of 3H-thymidine incorporation into DNA by greater than 90% in confluent cultures of human foreskin fibroblasts.

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L1 3083 FILE MEDLINE
L2 11344 FILE HCAPLUS

TOTAL FOR ALL FILES
L3 14427 CR1 OR CR2 OR DAF OR MCP OR C4BP

=> s (complement receptor 1) or (complement receptor 2) or (decay accelerating factor) or (membrane cofactor protein) or (c4# binding protein)
L4 1048 FILE MEDLINE
L5 1009 FILE HCAPLUS

TOTAL FOR ALL FILES
L6 2055 (COMPLEMENT RECEPTOR 1) OR (COMPLEMENT RECEPTOR 2) OR (DEC
AY ACCELERATING FACTOR) OR (MEMBRANE COFACTOR PROTEIN) OR
(C4# BINDING PROTEIN)

=> s l3 not l6
L7 2479 FILE MEDLINE
L8 10582 FILE HCAPLUS

TOTAL FOR ALL FILES
L9 13061 L3 NOT L6

=> s l9 and complement
L10 878 FILE MEDLINE
L11 735 FILE HCAPLUS

TOTAL FOR ALL FILES
L12 1613 L9 AND COMPLEMENT

=> s hybrid? or chimer? or fusion or truncat?
L13 306562 FILE MEDLINE
L14 279854 FILE HCAPLUS

TOTAL FOR ALL FILES
L15 586416 HYBRID? OR CHIMER? OR FUSION OR TRUNCAT?

=> s l12 and l15
L16 88 FILE MEDLINE
L17 52 FILE HCAPLUS

TOTAL FOR ALL FILES
L18 140 L12 AND L15

=> s l18 and py>1991
L19 39 FILE MEDLINE
L20 25 FILE HCAPLUS

TOTAL FOR ALL FILES
L21 64 L18 AND PY>1991

=> s l18 not l21
L22 49 FILE MEDLINE
L23 27 FILE HCAPLUS

TOTAL FOR ALL FILES
L24 76 L18 NOT L21

=> dup rem l24
PROCESSING COMPLETED FOR L24
L25 54 DUP REM L24 (22 DUPLICATES REMOVED)

=> s hybrid or chimer? or fusion or truncat?
L26 224142 FILE MEDLINE
L27 195916 FILE HCAPLUS

TOTAL FOR ALL FILES
L28 420058 HYBRID OR CHIMER? OR FUSION OR TRUNCAT?

=> s l12 and l28
L29 67 FILE MEDLINE
L30 39 FILE HCAPLUS

TOTAL FOR ALL FILES
L31 106 L12 AND L28

=> s l31 and py>1991
L32 33 FILE MEDLINE
L33 21 FILE HCAPLUS

TOTAL FOR ALL FILES
L34 54 L31 AND PY>1991

=> s l31 not l34
L35 34 FILE MEDLINE
L36 18 FILE HCAPLUS

TOTAL FOR ALL FILES
L37 52 L31 NOT L34

=> dup rem l37
PROCESSING COMPLETED FOR L37
L38 37 DUP REM L37 (15 DUPLICATES REMOVED)

=> d bib ab 1-

L38 ANSWER 1 OF 37 HCAPLUS COPYRIGHT 1997 ACS
AN 1991:490573 HCAPLUS
DN 115:90573
TI ***CR2*** receptor ligand compositions and methods for modulating immune cell functions
IN Lernhardt, Waldemar
PA California Institute of Biological Research, USA
SO PCT Int. Appl., 128 pp.
CODEN: PIXXD2
PI WO 9103251 A1 910321
DS W: CA, JP
RW: AT, BE, CH, DE, DK, ES, FR, GB, IT, LU, NL, SE
AI WO 90-US5027 900904
PRAI US 89-404679 890908
US 90-512118 900420
DT Patent
LA English
AB New ligands that bind the ***CR2*** receptor and modulate ***CR2*** -mediated events occurring in normal and neoplastic B-lymphocytes have been discovered. One set of these ligands is from the interferon .alpha. (IFN.alpha.) family. Synthetic polypeptides corresponding to the ***CR2*** receptor binding site present on a ***CR2*** ligand are disclosed together with polypeptide aggregates contg. the binding sites, therapeutic compns. contg. ***CR2*** ligands, methods to stimulate or inhibit B-lymphocyte proliferation and to inhibit Epstein-Barr virus infection in B-lymphocytes, antibodies, and methods of prepg. and using the polypeptides and antibodies. Amino acid and nucleotide sequences are present for a ***CR2*** ligand, IFN.alpha. strain A and IFN88, and an alk. phosphatase-IFN.alpha. ***fusion*** protein. A ***CR2*** ligand contg. 700 amino acid residues was encoded by a .lambda. 9-4 clone obtained from a Raji cDNA library.

L38 ANSWER 2 OF 37 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:51529 HCAPLUS
DN 116:51529
TI Extracellular domain of ***CR2*** glycoprotein in compositions and methods for detection and treatment of Epstein-Barr virus infection and immune disorders
IN Moore, Margaret D.; Cooper, Neil D.; Nemerow, Glen R.
PA Scripps Clinic and Research Foundation, USA
SO Can. Pat. Appl., 52 pp.
CODEN: CPXXEB
PI CA 2023779 AA 910224
AI CA 90-2023779 900822
PRAI US 89-398224 890823
DT Patent
LA English
AB Polypeptides of the B-lymphocyte membrane receptor glycoprotein ***CR2*** are disclosed together with polypeptide aggregates, compns., and methods for the treatment and detection of Epstein-Barr virus (EBV) infection and immunol. disorders involving ***CR2*** . CDNA coding for ***CR2*** was isolated and ***CR2*** /Baculovirus expression vectors were constructed for ***CR2*** polypeptides prodn. Preincubation of EBV with rCR2 (a recombinant ***CR2*** contg. residues 1-1005 of ***CR2***) resulted in dose-dependent inhibition of EBV infection of B-cells. When varying amts. of EBV were preincubated with a const. amt. of ***CR2*** (10 .mu.g) prior to addn. of B-cells, EBV induction of B-cell transformation was reduced by .apprx.90%.

L38 ANSWER 3 OF 37 MEDLINE DUPLICATE 1
AN 91239566 MEDLINE
TI Sites within the ***complement*** C3b/C4b receptor important for the specificity of ligand binding.
AU Krych M; Hourcade D; Atkinson J P
CS Howard Hughes Medical Institute, Washington University School of Medicine, St. Louis, MO 63110..
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES
OF AMERICA, (1991 May 15) 88 (10) 4353-7.
Journal code: PV3. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9108
AB Cysteine-rich repeated units of 40-70 amino acids are building blocks of many mammalian proteins, including 12 proteins of the ***complement*** system. Human ***complement*** arranged motifs, designated short consensus repeats (SCRs), which constitute the entire extracellular portion of this protein. Klickstein et al. [Klickstein, L. B., Bartow, T. J., Miletic, V., Rabson, L. D., Smith, J. A. & Fearon, D. T. (1988) J. Exp. Med. 168, 1699-1717 (abstr.)] localized a C4b binding domain to SCR-1 and/or SCR-2 and a C3b binding domain to SCR-8 and/or SCR-9. These SCRs bind different ligands, although SCR-1 and SCR-8 are 55% homologous and SCR-2 and SCR-9 are 70% homologous. To examine if one or two SCRs are required for ligand binding and to define sites within the SCRs that determine specificity of binding, mutagenesis analysis of a ***truncated*** , secreted form of ***CR1*** , called ***CR1*** -4 by Hourcade et al. [Hourcade, D., Meisner, D. R., Atkinson, J. P. & Holers, V. M. (1988) J. Exp. Med. 168, 1255-1270], was undertaken. The latter, composed of the first eight and one-half amino-terminal SCRs of ***CR1*** , efficiently bound C4b but not iC3. SCR-1 and SCR-2 were necessary for this interaction. Analysis of the mutant ***CR1*** -4 proteins, in which amino acids in SCR-1 and SCR-2 were substituted a few at a time with the homologous amino acids of SCR-8 and SCR-9, led to the identification of one amino acid in SCR-1 and three amino acids in SCR-2 important for C4b binding. Furthermore, five amino acids at the end of SCR-9, if placed in the homologous positions of SCR-2, conferred iC3 binding and are likely essential for ligand binding activity of SCR-8 and SCR-9. This iC3 binding occurred only if SCR-1 was present, indicating that two contiguous SCRs are necessary for this interaction. These results provide identification of amino acids within SCRs that are important for ligand binding.

L38 ANSWER 4 OF 37 MEDLINE DUPLICATE 2
AN 92043737 MEDLINE
TI CRRP: a guinea pig protein, identified by sequence homology to human ***CR1*** , which contains two short consensus repeat motifs and appears not to be transmembrane or secreted.
AU Moore F D Jr
CS Department of Surgery, Brigham and Women's Hospital, Harvard Medical School, Boston, MA 02115..
NC AI24139 (NIAID)
SO JOURNAL OF IMMUNOLOGY, (1991 Nov 15) 147 (10) 3615-22.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
OS GENBANK-M77760
EM 9202
AB cDNA from the C4b-binding site of the human C3b/C4b receptor (***CR1***) was used to find homologous sequences in the guinea pig. This cDNA identified an 18S mRNA species in guinea pig spleen, but not liver. Probing of a guinea pig spleen cDNA library identified clones with identical 1.5-kb inserts, which also hybridized to mRNA in spleen, but not liver. Sequence analysis of the insert revealed a single long open-reading frame coding for a 20,000 Mr protein consisting of two short consensus repeat motifs homologous to human ***CR1*** , and unique sequence at the amino- and carboxy-terminals of the short consensus repeats. This sequence did not encode peptides with features of transmembrane domains or signal peptides. Antibody to this ***complement*** receptor-related protein-beta galactosidase ***fusion*** protein recognized a 20,000 Mr protein in SDS lysates of guinea pig spleen, lymph node, lymphocytes, neutrophils, and peritoneal macrophages. Immunoprecipitation of human serum by this antibody revealed an 180,000 Mr protein reacting both with the anti-guinea pig protein antibody and with anti-human ***CR1*** antibody. Immunoprecipitation of guinea pig serum revealed no protein reacting with the anti-guinea pig protein antibody. Tissue staining of cultured peritoneal macrophages with this antibody showed intracellular staining, as opposed to membrane staining obtained with anti-guinea pig Ig antibody. The lack of membrane expression was confirmed by surface protein radiolabeling experiments and by fluorescent staining of surface proteins. Thus, we have identified a guinea pig protein with homology to human ***CR1*** , which may have an unusual property for this class of proteins in that it appears to be intracellular.

L38 ANSWER 5 OF 37 MEDLINE DUPLICATE 3
AN 91251206 MEDLINE
TI Inhibition of Epstein-Barr virus infection in vitro and in vivo by soluble ***CR2*** (CD21) containing two short consensus repeats.
AU Moore M D; Cannon M J; Sewall A; Finlayson M; Okimoto M; Nemerow G R
CS Department of Immunology, Scripps Clinic and Research Foundation, La Jolla, California 92037..
NC CA 36204 (NCI)
CA 52241 (NCI)
M01 RR00833 (NCRR)
SO JOURNAL OF VIROLOGY, (1991 Jul) 65 (7) 3559-65.
Journal code: KCV. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9109
AB The extracellular domain of ***CR2*** , the Epstein-Barr virus (EBV)/C3d receptor of B lymphocytes, contains 15 or 16 tandemly arranged short consensus repeat elements (SCR). Recombinant ***CR2*** proteins containing SCR 1 and 2 fused to Staphylococcus aureus protein A (PA- ***CR2***) and to murine ***complement*** factor H SCR 20 (CR2FH) were expressed in Escherichia coli and in insect cells, respectively. These recombinant ***CR2*** molecules retained functional activity as indicated by their ability to bind to C3dg in an enzyme-linked immunosorbent assay and to inhibit EBV gp350/220 binding to B cells. PA- ***CR2*** and CR2FH were as efficient in blocking EBV gp350/220 binding as the full-length ***CR2*** extracellular domain, indicating that the

first two SCR of ***CR2*** contain the majority of the ligand binding activity of the receptor. PA- ***CR2*** and CR2FH inhibited EBV-induced B-cell proliferation in vitro and blocked the development of EBV-induced lymphoproliferative disease in severe combined immunodeficient mice reconstituted with human lymphocytes. These studies indicate that soluble forms of ***truncated*** ***CR2*** proteins may have potential therapeutic value in the treatment of EBV-induced lymphoproliferative disorders in humans that involve viral replication.

L38 ANSWER 6 OF 37 HCAPLUS COPYRIGHT 1997 ACS
AN 1992:57213 HCAPLUS
DN 116:57213
TI B cell phenotype-dependent expression of the Epstein-Barr virus nuclear antigens EBNA-2 to EBNA-6: studies with somatic cell hybrids
AU Contreras-Brodin, Bertha A.; Anvret, Maria; Imreh, Stefan; Altiok, Ender; Klein, George; Masucci, Maria G.
CS Dep. Tumor Biol., Karolinska Inst., Stockholm, S-104 01, Swed.
SO J. Gen. Virol. (1991), 72(12), 3025-33
CODEN: JGVIAY; ISSN: 0022-1317
DT Journal
LA English
AB The expression of the transformation-assocd. Epstein-Barr virus (EBV)-encoded nuclear antigens (EBNAs) 1 to 6 and membrane protein LMP-1 was studied in a series of somatic cell hybrids derived from the ***fusion*** of the EBV-transformed lymphoblastoid cell line (LCL) KR-4, and the EBV-carrying Burkitt's lymphoma lines Daudi, P3HR-1 and Raji, with non-B cell lines of fibroblast, erythroid, myeloid and epithelial origin. Expression of EBNAs 2 to 6 was down-regulated in the hybrids in parallel with extinction of the B cell markers CD19, CD20, CD21, CD23, HLA class II, and surface or cytoplasmic Ig. LMP-1 was expressed independently of EBNA-2 in hybrids derived by the ***fusion*** of the LMP-1-pos. KR-4 and P3HR-1 cell lines with epithelial and myeloid cells, resp. LMP-1 was down-regulated in hybrids derived by the ***fusion*** of P3hR-1 with an erythroid cell line and in the ***hybrid*** between Raji and a mouse fibrosarcoma line. EBNA-1 was the only EBV antigen that was regularly expressed in the hybrids regardless of the dominating cellular phenotype. The autonomous expression of EBNA-1 suggests that its regulatory pathway is independent of phenotype-assocd. cellular or viral factors. In contrast, the expression of EBNAs 2 to 6 appears to require a B cell environment. EBNA-2 was shown to contribute to the regulation of LMP expression in B cells. Thus, LCL-carcinoma hybrids, the dominating epithelial phenotype is permissive for LMP expression in the absence of EBNA-2.

L38 ANSWER 7 OF 37 MEDLINE DUPLICATE 4
AN 92078855 MEDLINE
TI Mapping of the C3b-binding site of ***CR1*** and construction of a (***CR1***)2-F(ab')2 ***chimeric*** ***complement*** inhibitor.
AU Kalli K R; Hsu P H; Bartow T J; Ahearn J M; Matsumoto A K; Klickstein L B; Fearon D T
CS Graduate Program in Immunology, Johns Hopkins University School of Medicine, Baltimore, Maryland..
NC AI-22833 (NIAID)
AI-28191 (NIAID)
AI-07247 (NIAID)
+
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1991 Dec 1) 174 (6) 1451-60.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9203
AB ***CR1*** / ***CR2*** ***chimeric*** receptors in which various short consensus repeats (SCRs) of ***CR1*** were attached to ***CR2*** were transiently expressed on COS cells, and assessed for the binding of polymerized C3b (pC3b) and anti-***CR2*** by immunofluorescence. Of COS cells expressing ***chimeras*** containing SCR 1-4, 1-3, 2-4, 1-2, and 2-3 of the long homologous repeats (LHRs) -B or -C, 96%, 66%, 23%, 0%, and 0%, respectively, bound pC3b. K562 cells were stably transfected with wild-type ***CR1*** , deletion mutants of ***CR1*** , and the ***CR1*** / ***CR2*** ***chimeras*** , respectively, and assayed for binding of 125I-pC3b. The dissociation constants (Kd) for pC3b of wild-type ***CR1*** and the LHR-BD and -CD constructs were in the range of 1.0-2.7 nM, and of the ***CR1*** / ***CR2*** ***chimeras*** containing SCRs 1-4, 1-3, and 2-4 of LHR-B or -C were 1.8-2.4, 6-9, and 22-36 nM, respectively. The factor I-cofactor function of the ***CR1*** / ***CR2*** ***chimeras*** paralleled the C3b-binding function of the constructs. A ***CR1*** /immunoglobulin (Ig) ***chimeric*** protein was prepared by fusing SCRs 1-4 of LHR-B to the heavy chains of a murine F(ab')2 anti-nitrophenacetyl (NP) monoclonal antibody. The (***CR1***)2-F(ab')2 ***chimera*** , which retained its specificity for NP, was as effective as soluble, full-length ***CR1*** in binding pC3b, serving as a cofactor for factor I-mediated cleavage of C3b, and inhibiting activation of the alternative pathway, indicating that the bivalent expression of these SCRs reconstitutes the alternative pathway inhibitory function of ***CR1*** . The feasibility of creating ***CR1*** /Ig ***chimeras*** makes possible a new strategy of targeting ***complement*** inhibition by the use of Ig ***fusion*** partners having particular antigenic specificities.

L38 ANSWER 8 OF 37 MEDLINE
AN 92078839 MEDLINE
TI Determination of the structural basis for selective binding of Epstein-Barr virus to human ***complement*** receptor type 2.
AU Martin D R; Yuryev A; Kalli K R; Fearon D T; Ahearn J M
CS Graduate Program in Immunology, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205..
NC AI-22833 (NIAID)
AI-28191 (NIAID)
GM-43803 (NIGMS)
+
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1991 Dec 1) 174 (6) 1299-311.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9203
AB Epstein-Barr virus (EBV) is an oncogenic herpesvirus that selectively infects and immortalizes human B lymphocytes. One determinant of this narrow tropism is human ***CR2*** , the only viral receptor within the superfamily of proteins that contain short consensus repeats (SCRs). Human ***CR2*** serves as a receptor for both C3dg and the gp350/220 glycoprotein of EBV, and binds the monoclonal antibody (mAb) OKB7, which blocks binding of both ligands to the receptor. In contrast, although murine ***CR2*** is capable of binding human C3dg and this interaction can be blocked with the mAb 7G6, it does not bind OKB7 or EBV. We have determined the structural basis for absolute specificity of EBV for human ***CR2*** through characterization of a panel of 24 human-murine ***chimeric*** receptors, all of which bind human C3dg. The results indicate that preferential binding of EBV to human ***CR2*** is not due to unique amino acids that are capable of binding the virus, but reflects a distinct receptor conformation that can be achieved in murine ***CR2*** with single amino acid substitutions in two discontinuous regions of the primary structure: replacement of proline at position 15 with the corresponding serine from human ***CR2*** , and elimination of a potential N-linked glycosylation site between SCR-1 and SCR-2. Furthermore, species-specific binding of EBV, OKB7, and 7G6 can all be manipulated through substitutions among residues 8-15, suggesting that this octapeptide is part of a structural determinant that is critical for binding of both viral and natural ligands to ***CR2*** .

L38 ANSWER 9 OF 37 MEDLINE DUPLICATE 5
AN 92049739 MEDLINE
TI Complementation of transforming domains in E1a/myc chimaeras.
AU Ralston R
CS Chiron Corporation, Emeryville, California 94608..
SO NATURE, (1991 Oct 31) 353 (6347) 866-8.
Journal code: NSC. ISSN: 0028-0836.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9202
AB The myc oncogene is functionally similar to adenovirus E1a in its ability to collaborate with activated ras oncogenes to transform primary fibroblasts. The transforming functions of E1a and myc have been mapped to two distinct regions in each protein. I investigated the functional similarities between E1a and myc by constructing E1a/myc chimaeras to discover whether the individual transforming domains of E1a could ***complement*** individual myc-transforming domains. Transformation assays in rat embryo fibroblasts demonstrated that the N-terminal transforming domain of E1a (***CR1***) could ***complement*** the C-terminal transforming domain of myc in cis, and that the reciprocal chimaera (N-terminal myc/C-terminal E1a) was also active. Chimaeras constructed using domains from transformation-defective mutants of either E1a or myc were inactive, indicating that both E1a and myc domains contribute to function. These experiments suggest that transformation by myc and E1a may involve interactions with common substrates.

L38 ANSWER 10 OF 37 MEDLINE DUPLICATE 6
AN 91302808 MEDLINE
TI Interaction of iC3b with recombinant isotypic and ***chimeric*** forms of ***CR2*** .
AU Kalli K R; Ahearn J M; Fearon D T
CS Graduate Program in Immunology, Johns Hopkins University School of Medicine, Baltimore, MD 21205..
NC AI28191 (NIAID)
AI22833 (NIAID)
AI07247 (NIAID)
+

SO JOURNAL OF IMMUNOLOGY, (1991 Jul 15) 147 (2) 590-4.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 9110
AB ***CR2*** is a component of a signal transduction complex on B lymphocytes that augments B cell responses to Ag. We have quantitatively assessed binding by the two isotypic forms of ***CR2*** for two of its ligands, the polymerized iC3b (p(iC3b)) fragment of C3, and gp350/220, the EBV membrane protein. The recombinant 15-SCR or 16-SCR forms of ***CR2*** bound p(iC3b) with identical affinities. Full binding activity of ***CR2*** for p(iC3b) was observed with a ***chimera*** comprised of SCR-1 and -2 of ***CR2*** fused to SCR-17 through -30 of ***CR1*** . Therefore, the alternatively spliced SCR-10a has no role in binding p(iC3b), and the binding activity of wild type receptor for iC3b can be reconstituted with SCR-1 and -2 of ***CR2*** . The binding affinities of the two isoforms of ***CR2*** for soluble gp350/220 were also similar. Additional sites in the C3c region of C3 have been postulated also to interact with ***CR2*** . However, monomeric iC3b and C3d were equally effective in inhibiting the binding of p(iC3b) to ***CR2*** , indicating that the C3c region of iC3b does not contribute to the interaction of iC3b with ***CR2*** . Finally, the relative abilities of C3b and iC3b to bind to ***CR1*** and ***CR2*** were compared. The conversion of C3b to iC3b generated a ligand with an approximate 100-fold decrease in affinity for ***CR1*** and a 10-fold increased affinity for ***CR2*** , resulting in a 1000-fold greater likelihood for binding to the latter receptor that may then promote B cell activation.

L38 ANSWER 11 OF 37 MEDLINE
AN 92127740 MEDLINE
TI Intraabdominal sepsis: enhanced autooxidative effect on polymorphonuclear leukocyte cell surface receptor expression.
AU Simms H H; D'Amico R; Burchard K W
CS Department of Surgery, Rhode Island Hospital, Providence, RI 02903..
SO CIRCULATORY SHOCK, (1991 Aug) 34 (4) 356-63.
Journal code: C9Y. ISSN: 0092-6213.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9205
AB We investigated the effects of untreated intraabdominal sepsis on the interrelationship between PMN oxidative metabolism and cell surface receptor expression. Female swine underwent either sham laparotomy (n = 7) or cecal ligation and incision (n = 9) with assays conducted on postoperative days (POD) 0, 1, 4, and 8. Superoxide anion production, intracellular H2O2 production, and the cell surface expression of Fc gamma RII, III, ***CR1*** , and CR3 were measured. In addition, phagocytosis of serum-opsonized zymosan was used as a multivalent ligand for CR3 and subsequently Fc gamma RII, III, and ***CR1*** expression were assayed to determine if intraabdominal sepsis induces a linkage between ***complement*** and Fc gamma receptor expression. Superoxide anion production increased between POD 0 and 4 and fell between POD 4 and 8 in animals with untreated intraabdominal sepsis. Intracellular H2O2 production rose between POD 0 and 1 and then fell progressively in animals with untreated intraabdominal sepsis. Simulation of the oxidative burst using glucose/glucose oxidase reduced Fc gamma RII and III expression in both sets of animals with a greater reduction seen by POD 4 in animals with intraabdominal sepsis. ***CR1*** /CR3 expression was increased with glucose/glucose oxidase by POD 4 in the presence of intraabdominal sepsis. Xanthine/xanthine oxidase did not alter cell surface receptor expression. Phagocytosis of serum-opsonized zymosan decreased subsequent Fc gamma RII expression in animals with intraabdominal sepsis by POD 4.(ABSTRACT ***TRUNCATED*** AT 250 WORDS)

L38 ANSWER 12 OF 37 MEDLINE DUPLICATE 7
AN 91079556 MEDLINE
TI Recombinant soluble human ***complement*** receptor type 1 inhibits inflammation in the reversed passive arthus reaction in rats.
AU Yeh C G; Marsh H C Jr; Carson G R; Berman L; Concino M F; Scesney S M; Kuestner R E; Skibbens R; Donahue K A; Ip S H
CS T Cell Sciences, Inc., Cambridge, MA 02139..
SO JOURNAL OF IMMUNOLOGY, (1991 Jan 1) 146 (1) 250-6.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 9104
AB The human ***CR1*** was genetically engineered by site directed mutagenesis into a ***truncated*** form which was secreted from transfected Chinese hamster ovary cells. This soluble recombinant ***CR1*** (sCR1) was purified from the supernatants of the Chinese hamster ovary cells cultured in a hollow fiber bioreactor. sCR1 inhibits the C3 and C5 convertases of the classical and the alternative pathways in vitro. The ability of sCR1 to inhibit the immune complex-mediated inflammation in vivo was tested in a rat reversed passive Arthus reaction model. Administration of sCR1 at the dermal sites reduced the Arthus vasculitis in a dose-dependent manner as judged by both gross and microscopic examination, as well as by immunohistologic localization of C3 and C5b-9 neoantigen deposits. These data suggest that sCR1 inhibits the Arthus reaction by interrupting the activation of the C cascade, hence limiting the detrimental immune complex-induced tissue damage in vivo.

L38 ANSWER 13 OF 37 MEDLINE DUPLICATE 8
AN 92022529 MEDLINE
TI Suppression of the immune response by a soluble ***complement*** receptor of B lymphocytes.
AU Hebell T; Ahearn J M; Fearon D T
CS Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, MD 21205..
NC AI-22833 (NIAID)
AI-28191 (NIAID)
GM-43803 (NIGMS)
SO SCIENCE, (1991 Oct 4) 254 (5028) 102-5.
Journal code: UJ7. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9201
AB The CD19- ***CR2*** complex of B lymphocytes contains proteins that participate in two host-defense systems, the immune and ***complement*** systems. The ligand for the subunit of the immune system, CD19, is not known, but the ***complement*** receptor subunit, ***CR2*** (CD21), binds activation fragments of the C3 component of the ***complement*** system and may mediate immunopotentiating effects of ***complement*** . A recombinant, soluble ***CR2*** was prepared by fusing the C3-binding region of the receptor to immunoglobulin G1 (IgG1). The (***CR2***)2-IgG1 ***chimera*** competed with cellular ***CR2*** for C3 binding and suppressed the antibody response to a T cell-dependent antigen when administered to mice at the time of immunization. This inhibitory effect of (***CR2***)2-IgG1 demonstrates the B cell-activating function of the CD19- ***CR2*** complex and suggests a new method for humoral immunosuppression.

L38 ANSWER 14 OF 37 MEDLINE DUPLICATE 9
AN 91199434 MEDLINE
TI The effect of antibody isotype and antigenic epitope density on the ***complement*** -fixing activity of immune complexes: a systematic study using chimaeric anti-NIP antibodies with human Fc regions.
AU Lucisano Valim Y M; Lachmann P J
CS Molecular Immunopathology Unit, Medical Research Council Centre, Cambridge, England..
SO CLINICAL AND EXPERIMENTAL IMMUNOLOGY, (1991 Apr) 84 (1) 1-8.
Journal code: DD7. ISSN: 0009-9104.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9107
AB A systematic study has been carried out to investigate the role of immunoglobulin isotype, epitope density, and antigen/antibody ratio on the capacity of immune complexes to activate the classical and alternative pathways of human ***complement*** and for the complexes subsequently to bind to erythrocyte C3b-C4b receptors (CRI). For this purpose, a series of chimaeric monoclonal anti-NIP antibodies was used, which all shared the same combining site but had different human constant domains. Antigen epitope density was varied by coupling different numbers of NIP hapten molecules to bovine serum albumin. All three parameters affect ***complement*** fixation. In general, ***complement*** activation is better in antibody excess and at equivalence than it is in antigen excess, and better at high epitope density than at low epitope density, although the effects are variable for different immunoglobulin isotypes and for the two pathways. It has been confirmed that IgG1 and IgG3 are good activators of the classical pathway and are tolerant to variations in both epitope density and antigen/antibody ratio. IgG4 and IgA do not activate the classical pathway in any circumstances. IgG2 activates the classical pathway only at high epitope density and at equivalence or antibody excess. IgM activates the classical pathway well only at the higher epitope densities and at equivalence or antibody excess but, in addition, shows an interesting and unexpected prozone phenomenon where immune complex in antibody excess inhibits ***complement*** activation by the classical pathway. The results of the alternative pathway activation are strikingly different. IgA is by far the best activator of the alternative pathway and is relatively tolerant to epitope density and to antigen/antibody ratio. IgM, IgG1 and IgG3 do not significantly activate the alternative pathway in any circumstances. IgG2 is the best IgG subclass for alternative pathway activation but requires high epitope density and equivalence or antibody excess. Binding to ***CR1*** in general parallels the amount of ***complement*** fixed independent to the pathway by which it is fixed. However, IgG1 and IgG3 complexes in antigen excess activate ***complement*** well but bind poorly to ***CR1*** . Nascently formed complexes seem to bind ***complement*** in a way that is

similar to that bound by preformed complexes, but are then less able to bind to red cell ***CR1*** . These observations help to explain the pathogenesis of ***complement*** activation in various autoimmune and immune complex diseases such as systemic lupus erythematosus, autoimmune thyroiditis and others.

L38 ANSWER 15 OF 37 MEDLINE DUPLICATE 10
AN 90324211 MEDLINE
TI Structural requirements for C3d,g/Epstein-Barr virus receptor (***CR2*** /CD21) ligand binding, internalization, and viral infection.
AU Carel J C; Myones B L; Frazier B; Holers V M
CS Howard Hughes Medical Institute Laboratories, Washington University School of Medicine, St. Louis, Missouri 63110..
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1990 Jul 25) 265 (21) 12293-9. Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9011
AB The structure of ***CR2*** , the human C3d,g/EBV receptor (***CR2*** /CD21) consists of 15 or 16 60-70 amino acid repeats called short consensus repeats (SCRs) followed by a transmembrane and a 34-amino acid intracytoplasmic domain. Functions of ***CR2*** include binding the human ***complement*** component C3d,g when it is covalently attached to targets or cross-linked in the fluid phase. In addition, ***CR2*** binds the Epstein-Barr virus (EBV) and mediates internalization of EBV and subsequent infection of cells. In order to explore functional roles of the repetitive extracytoplasmic SCR structure and the intracytoplasmic domain of ***CR2*** , we have created ***truncated*** ***CR2*** (rCR2) mutants bearing serial deletions of extracytoplasmic SCRs and also the intracytoplasmic tail. We then stably transfected these rCR2 mutants into two cell lines, murine fibroblast L cells and human erythroleukemic K562 cells. Phenotypic analysis of these expressed mutants revealed that 1) The C3d,g- and EBV-binding sites are found in the two amino-terminal SCRs of ***CR2*** , 2) expression of SCRs 3 and 4 is further required for high affinity binding to soluble cross-linked C3d,g, 3) the intracytoplasmic domain of ***CR2*** is not required for binding C3d,g or EBV but is necessary for internalization of cross-linked C3d,g as well as for EBV infection of cells, 4) monoclonal anti- ***CR2*** antibodies with similar activities react with single widely separated epitopes, and 5) no functional roles can yet be clearly assigned to SCRs 5-15, as rCR2 mutants not containing these SCRs show no major differences from wild-type rCR2 in binding or internalizing cross-linked C3d,g or mediating EBV binding and infection.

L38 ANSWER 16 OF 37 MEDLINE
AN 91079504 MEDLINE
TI Evaluation of the mechanisms responsible for the reduction in erythrocyte ***complement*** receptors when immune complexes form in vivo in primates.
AU Cosio F G; Shen X P; Birmingham D J; Van Aman M; Hebert L A
CS Department of Internal Medicine, Ohio State University, Columbus 43210..
NC HL25404 (NHLBI)
SO JOURNAL OF IMMUNOLOGY, (1990 Dec 15) 145 (12) 4198-206. Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 9104
AB Patients with immune complex-(IC) mediated diseases frequently have low levels of ***CR1*** on E. The present study was undertaken to determine the role of circulating IC in causing low E- ***CR1*** levels. E- ***CR1*** were enumerated by measuring the binding of anti- ***CR1*** mAb (E11) and rabbit anti- ***CR1*** antibodies (RbaCR1) to E. In addition, the distribution of ***CR1*** among E was assessed by flow cytometry of E stained with E11 and RbaCR1 and by evaluating the binding of E11-coated fluorescent beads (E11-beads) to E. E11-beads bind to clusters of ***CR1*** on E. Five cynomolgus monkeys (CYN) were preimmunized to bovine gamma-globulin (BGG). E- ***CR1*** changes in these animals were assessed: 1) acutely, during the first 60 min after an infusion of BGG and 2) chronically, during daily administration of BGG infusions over 2 wk. Acutely, there was a decrease in the number of E- ***CR1*** as measured by E11 binding to E (E11/ ***CR1***). This decrease was not attributable to occupancy of ***CR1*** by IC because the decrease in E11/ ***CR1*** number persisted after the IC had been cleared from E. By comparing the E11/ ***CR1*** levels in arterial blood to hepatic vein blood (n = 5), or in pulmonary artery blood (n = 1), we determined that the acute decrease in E11/ ***CR1*** number did not occur whereas E circulated through liver, spleen, or lung. The decrease in E11/ ***CR1*** number required the binding of IC to E because it did not occur after BGG was infused into nonimmunized CYN (n = 2) or into a preimmunized ***complement*** -depleted CYN. The decrease in E11/ ***CR1*** number was not due to loss of ***CR1*** from E because E11/ ***CR1*** number recovered 24 h after infusion of BGG and in addition, enumeration of E- ***CR1*** with RbaCR1 and E11-beads did not reflect a decrease in E- ***CR1*** number. After several daily BGG infusions there was a persistent decrease in E- ***CR1*** levels and that decrease appeared to be mainly the result of loss of ***CR1*** from E because the decrease was confirmed with all methods of E- ***CR1*** measurement and because E- ***CR1*** levels recovered only slowly after BGG infusions were discontinued. Both in vitro and in vivo IC bound preferentially to subpopulations of E, identified by their ability to bind multiple E11-beads and by their high intensity staining with the anti- ***CR1*** antibodies E11 and RbaCR1.(ABSTRACT ***TRUNCATED*** AT 400 WORDS)

L38 ANSWER 17 OF 37 MEDLINE DUPLICATE 11
AN 91010789 MEDLINE
TI A molecular and immunochemical characterization of mouse ***CR2*** . Evidence for a single gene model of mouse ***complement*** receptors 1 and 2.
AU Molina H; Kinoshita T; Inoue K; Carel J C; Holers V M
CS Howard Hughes Medical Institute Laboratories, Washington University School of Medicine, St. Louis, MO 63110..
SO JOURNAL OF IMMUNOLOGY, (1990 Nov 1) 145 (9) 2974-83. Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
OS GENBANK-M61132
EM 9101
AB The relationships between functional, biochemical, and genetic homologues of human and mouse C receptors 1 (***CR1***) and 2 (***CR2***) are incompletely understood. We have isolated and characterized a partial mouse ***CR2*** cDNA clone and determined the exon-intron organization of the gene encoding it. Together they predict a form of mouse ***CR2*** highly identical to the 15 short consensus repeat form of human ***CR2*** . Strong similarities in genomic organization and exon-intron junctions indicate that this mouse gene and human ***CR2*** are evolutionary homologues. A polyclonal rabbit anti-mouse ***CR2*** ***fusion*** protein, BRN-1, was prepared. BRN-1 immunoprecipitates bands of 155 to 160 kDa under nonreducing conditions in mouse ***CR2*** expressing B cell lines. In mouse spleen a doublet of 155 kDa and 190 kDa under nonreducing and 165 and 205 kDa under reducing conditions is recognized by immunoprecipitation and Western blot analysis. Staphylococcus aureus V8 protease maps of these two proteins show many shared bands. Crossed immunoprecipitation using BRN-1 and 7E9, a previously described mAb reported to identify the 190-kDa mouse ***CR1*** and a smaller 150-kDa protein, indicates that both antibodies react with the same proteins. Therefore, by using BRN-1 we have now linked the genetic mouse ***CR2*** to its functional, biochemically characterized gene product. The observation that BRN-1 also recognizes a second 190-kDa mouse protein defined functionally as a homologue of human ***CR1*** , and that these proteins have very similar peptide maps, provides strong evidence that these two proteins are expressed by a single mouse ***CR2*** / ***CR1*** transcription unit.

L38 ANSWER 18 OF 37 MEDLINE
AN 90355258 MEDLINE
TI Untreated intra-abdominal sepsis: lack of synergism between polymorphonuclear leukocyte (PMN) ***complement*** receptors ***CR1*** /CR3 and IgG receptor FcRIII.
AU Simms H H; D'Amico R; Burchard K
CS Department of Surgery, Rhode Island Hospital, Providence 02903..
SO JOURNAL OF TRAUMA, (1990 Aug) 30 (8) 1027-31. Journal code: KAF. ISSN: 0022-5282.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 9011
AB We have examined the effects of untreated intra-abdominal sepsis on polymorphonuclear leukocyte (PMN) phagocytosis. Phagocytosis was studied in a receptor-specific fashion looking at the interrelationship between FcRIII-, ***complement*** receptor (***CR1***)-, and ***complement*** receptor 3 (CR3)-mediated phagocytosis. Twelve swine underwent either sham laparotomy (n = 5) or laparotomy with cecal ligation and incision (n = 7) to induce intra-abdominal sepsis. PMN phagocytosis was assayed on POD 1, 4, and 8. In animals undergoing sham laparotomy, FcRIII-mediated phagocytosis was less than 10% on all days and was augmented with the addition of C3b or C3bi to the target particles (FcR + ***CR1*** or FcR + CR3 greater than FcR alone). In animals undergoing cecal ligation and incision, baseline FcRIII-mediated phagocytosis increased between POD 1 and 4 and fell between POD 4 and 8. No increase in phagocytosis was seen on POD 4 or 8 with the addition of C3b or C3bi to the target particles (FcR + ***CR1*** or FcR + CR3 = FcR alone). Preligation of the FcRIII but not FcRII or FcRI receptor with a monoclonal antibody (3G8) markedly reduced phagocytosis in the animals with intraabdominal sepsis.(ABSTRACT ***TRUNCATED*** AT 250 WORDS)

L38 ANSWER 19 OF 37 MEDLINE
AN 91079800 MEDLINE
TI Altered erythrocyte ***CR1*** binding kinetics compensate for decreased binding capacity in rheumatoid arthritis.
AU Hargrove J K; Meryhew N L; Runquist O A
CS Department of Medicine, University of Minnesota Medical School, Minneapolis..
SO JOURNAL OF CLINICAL CHEMISTRY AND CLINICAL BIOCHEMISTRY, (1990 Aug) 28 (8) 533-41.
Journal code: I3U. ISSN: 0340-076X.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9104
AB Patients with rheumatoid arthritis have decreased numbers of ***CR1*** per erythrocyte and decreased binding of immune complexes to erythrocytes. Overall erythrocyte immune complex binding activity depends on both the number and the binding kinetics of ***CR1*** . We measured kinetic parameters for the interaction between a ***complement*** -containing dsDNA:anti-dsDNA probe and erythrocytes in patients with rheumatoid arthritis and normal controls. The results indicate that: 1) the maximum quantity of immune complexes bound per erythrocyte was significantly decreased in rheumatoid arthritis compared with normal controls (p less than or equal to 0.009); 2) the steady state binding constant, Kss, and the association rate constant for binding of immune complexes to erythrocytes, ka, were significantly increased in rheumatoid arthritis versus normal controls (p less than or equal to 0.0001 and 0.002 respectively); 3) the dissociation rate constant for the release of bound immune complexes from erythrocytes, kd, was slightly smaller in rheumatoid arthritis but this difference was not statistically significant; and 4) the energies of activation for the association and dissociation reactions, Eaa, and Ead, did not differ between the two groups. These data confirm that while the maximum quantity of immune complexes bound per erythrocyte is decreased in rheumatoid arthritis, the association rate constants are larger and dissociation rate constants slightly smaller than those of normal controls. Changes in these kinetic parameters compensate for the decrease in the maximum quantity of immune complexes bound per erythrocyte.(ABSTRACT ***TRUNCATED*** AT 250 WORDS)

L38 ANSWER 20 OF 37 MEDLINE
AN 90164551 MEDLINE
TI Comparison of peritoneal white blood cell parameters from continuous ambulatory peritoneal dialysis patients with a high or low incidence of peritonitis.
AU Holmes C J; Lewis S L; Kubey W Y; Van Epps D E
CS Baxter Healthcare, Round Lake, IL 60073..
SO AMERICAN JOURNAL OF KIDNEY DISEASES, (1990 Mar) 15 (3) 258-64.
Journal code: 3H5. ISSN: 0272-6386.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9005
AB The purpose of this study was to determine if there were differences in selected dialysate white blood cells (WBC) parameters between continuous ambulatory peritoneal dialysis (CAPD) patient groups identified as having a high or low incidence of peritonitis. Parameters studied were total peritoneal WBC yield, percentage and absolute number of various WBC types, and expression of WBC receptors known to be involved in normal host defense mechanisms. WBCs were obtained from peritoneal dialysis effluents (overnight dwell), which were collected at monthly intervals for 6 to 8 months from eight CAPD patients--four with a history of high peritonitis incidence (HPI) (more than two episodes in 12 months) and four with a history of low peritonitis incidence (LPI) (no episodes in more than 24 months). Our results demonstrated that there was no significant difference in the overall mean total cell yields or absolute cell counts between the two patient groups. WBC differentials, although differing somewhat among patients, stayed quite stable over time for an individual patient and there was no significant difference between the two patient groups. Analysis of receptors on the peritoneal WBC was performed using flow cytometry and fluorescein-conjugated chemotactic factors (C5a and fMet-Leu-Phe-Lys), as well as monoclonal antibodies specific for Fc receptors and ***complement*** receptors, ***CR1*** (CD35) and CR3 (CD11b). Although there was a trend toward increased expression of all these receptors in the HPI patients, there was no significant difference in the fluorescence intensity of peritoneal neutrophils or macrophages that expressed these receptors between the two patient groups.(ABSTRACT ***TRUNCATED*** AT 250 WORDS)

L38 ANSWER 21 OF 37 MEDLINE
AN 90370390 MEDLINE
TI Expression of the ***complement*** receptors ***CR1*** and CR3 and the type III Fc gamma receptor on neutrophils from newborn infants and from fetuses with Rh disease.
AU Smith J B; Campbell D E; Ludomirsky A; Polin R A; Douglas S D; Garty B Z; Harris M C
CS Department of Pediatrics, University of Pennsylvania School of Medicine, Philadelphia..
NC HL-27068 (NHLBI)
NS-17752 (NINDS)
SO PEDIATRIC RESEARCH, (1990 Aug) 28 (2) 120-6.
Journal code: OWL. ISSN: 0031-3998.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9012
AB Developmental defects in neutrophil function, including diminished expression of plasma membrane receptors, may play an important role in the susceptibility of the newborn infant to infection. We used monoclonal antibodies and flow cytometry to study the expression of ***complement*** receptor type one (***CR1***), ***complement*** receptor type three (CR3), and Fc gamma receptor type three (FcRIII) on neutrophils from six fetuses with Rh disease, 10 preterm infants, nine term infants, and nine adults. Expression of the ***complement*** receptors on unstimulated cells was similar for all groups, but significant differences in ***complement*** receptor expression were observed after stimulation with N-formyl-methionyl-leucyl-phenylalanine (FMLP). Fetal, preterm, and term infant neutrophils expressed less CR3 than FMLP-stimulated neutrophils of adults [61 +/- 2, 48 +/- 4, and 66 +/- 4% (mean +/- SEM) of the mean for adults, p less than 0.05]. FMLP-stimulated ***CR1*** expression for these groups was 61 +/- 6, 73 +/- 6, and 91 +/- 9% of the adult mean (p less than 0.05, fetal versus term infant and adult). Expression of both CR3 and ***CR1*** increased with postconceptional age in the infants (r2 = 0.49, p less than 0.001 for CR3; r2 = 0.23, p less than 0.05 for ***CR1***). Neutrophils of the preterm and term infants expressed less FcRIII than adult neutrophils (68 +/- 10 and 77 +/- 7% of the adult mean, p less than 0.05, for FMLP-stimulated cells), whereas fetal neutrophil FcRIII expression did not differ from that of the adult.(ABSTRACT ***TRUNCATED*** AT 250 WORDS)

L38 ANSWER 22 OF 37 MEDLINE
AN 90112003 MEDLINE
TI Expression of the C3d/EBV receptor and of other cell membrane surface markers is altered upon HIV-1 infection of myeloid, T, and B cells.
AU Larcher C; Schulz T F; Hofbauer J; Hengster P; Romani N; Wachter H; Dierich M P
CS Institut fur Hygiene, Universitat Innsbruck, Austria..
SO JOURNAL OF ACQUIRED IMMUNE DEFICIENCY SYNDROMES, (1990) 3 (2) 103-8.
Journal code: JOF. ISSN: 0894-9255.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9004
AB Human cell lines (the T-cell lines H9, Jurkat, and HUT102, the myeloid lines U937 and HL60, and the Raji B cell line) were infected with HIV-1. HIV-1 antigen could be detected by immunofluorescence analysis in more than 50% of T cells and myeloid cells 15 days after infection. Infection of Raji cells took more than 2-3 months. Studies of cell surface marker expression revealed remarkable changes after HIV-1 infection of Raji cells: expression of ***CR2*** (C3d/EBV receptor, CD19, CD20, CD22, CD23, CD10, and surface IgM) were highly reduced, in the case of ***CR2*** and membrane-IgM from 100 to 0%, whereas levels of CD37 and CD38 remained unaltered by HIV-1 infection. U937 cells showed a reduction of CD4 expression from 14 to 5% after HIV-1 infection; the CR3 expression slightly increased from 25 to 30%. In contrast, HLA-DR was only expressed (21%) after HIV-1 infection but not in uninfected U937 cells. Expression of HLA-DR could be detected also in HL60 cells (33%) after HIV-1 infection. In H9 cells, CD4 was reduced from 60 to 30% after HIV-1 infection, whereas HLA-DR and CD25/IL-2 receptor expression increased from 16 to 90% and from 0 to 50%, respectively. CD4 was reduced from 70 to 0% from Jurkat cells after HIV-1 infection, whereas expression of ***CR2*** was only slightly diminished from 8 to 4%. Expression of ***CR1*** and HLA-DR was slightly increased in these cells (1 to 3%).(ABSTRACT ***TRUNCATED*** AT 250 WORDS)

L38 ANSWER 23 OF 37 MEDLINE
AN 91077160 MEDLINE
TI Roles of the ***complement*** receptor type 1 (***CR1***) and type 3 (CR3) on phagocytosis and subsequent phagosome-lysosome ***fusion*** in Salmonella-infected murine macrophages.
AU Ishibashi Y; Arai T
CS Department of Microbiology, Meiji College of Pharmacy, Nozawa, Tokyo, Japan..
SO FEMS MICROBIOLOGY IMMUNOLOGY, (1990 Sep) 2 (2) 89-96.
Journal code: AO3. ISSN: 0920-8534.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9104
AB The receptors involved in the recognition of Salmonella typhimurium and S. typhi by murine macrophages were identified, and their

relevance to phagosome-lysosome ***fusion*** was also investigated. Phagocytosis of S. typhimurium by murine macrophages was dependent on the opsonization with normal fresh serum, although the opsonin had no triggering activity in phagosome-lysosome ***fusion*** . In contrast, the opsonization of S. typhi with normal fresh serum efficiently triggered both phagocytosis and following phagosome-lysosome ***fusion*** . Anti-murine ***CR1*** antibody suppressed phagocytosis of S. typhimurium by 36%, whereas anti-CR3 antibody, mannan, and advanced glycosylation endproducts (AGE)-BSA all failed to prevent phagocytosis of S. typhimurium, suggesting that ***CR1*** may only contribute to the recognition of S. typhimurium and may possibly play a minor role. Other receptors involved may also influence the outcome of phagocytosis in terms of phagosome-lysosome ***fusion*** . In the case of S. typhi, only anti-CR3 antibody significantly inhibited not only phagocytosis of S. typhi but also following phagosome-lysosome ***fusion*** . Treatment with K76COONa, an inhibitor of C3bINA (I factor), resulted in a marked inhibition of phagosome-lysosome ***fusion*** in S. typhi-infected macrophages, although no significant inhibition was observed on phagocytosis of S. typhi. These results suggest that S. typhimurium and S. typhi may be recognized at least in part by ***CR1*** and CR3, respectively, and that the recognition by CR3 but not ***CR1*** is functionally associated with subsequent phagosome-lysosome ***fusion*** in murine macrophages.

L38 ANSWER 24 OF 37 MEDLINE
AN 91223162 MEDLINE
TI ***Complement*** components and receptors: deficiencies and disease associations.
AU Bartholomew W R; Shanahan T C
CS Erie County Medical Center, Erie County Laboratory, Buffalo, New York..
SO IMMUNOLOGY SERIES, (1990) 52 33-51. Ref: 48
Journal code: A13. ISSN: 0092-6019.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 9108
AB The ***complement*** system, accessory to many immunological functions, consists of a number of interdependent components and receptors. Numerous in vitro approaches have elucidated the biological role of these components and receptors. However, it is the in vivo "natural" experiments that underscore their importance. The phagocytosis and subsequent digestion of pyogenic bacteria is significantly enhanced by the fixation of the third ***complement*** component to the bacterial cell wall. Equally important is the intact expression of a receptor (CR3) for the C3b cleavage fragment. Breakdown in this ligand-receptor interaction due to either C3 or CR3 deficiency leads to pyogenic infection. Interestingly, C3-deficient individuals do not demonstrate leukocytic infiltration at the site of infection. Undoubtedly, this is due to the lack of C5 convertase and failure to produce C5a. CR3-deficient individuals, on the other hand, do demonstrate leukocytosis since the third ***complement*** component is functional. C3 deficiency is not necessarily a primary lesion and may be secondary to factor I deficiency. In this case, the C3b fragment, along with factor B, acts as a C3 convertase. Inefficient inactivation of C3b, due to factor I deficiency, leads to the uncontrolled consumption of the third component, resulting in C3 deprivation. It appears that phagocytosis by neutrophils and monocytes followed by enzyme-interaction is not sufficient for destruction of the Neisseria organisms. In addition to this leukocyte activity, an intact membrane attack complex, composed of the late ***complement*** components C5, 6, 7, 8, and 9, is required for the lysis of these bacteria. This is supported by findings that individuals deficient in late components are highly susceptible to systemic Neisseria infections. Diseases of an autoimmune nature are frequently associated with a deficiency of one of the early ***complement*** components C1, C2, or C4 and a deficiency of erythrocytic ***CR1*** receptors as well. This may suggest that proper interaction between a ***complement*** fragment of the immune complex with the ***complement*** receptor expressed on the erythrocyte is important for proper management and clearance of the complex. Deficiency of the early ***complement*** components would prevent the activation of C3 and the fixation of a resulting C3 cleavage product. In this case, erythrocytes would be unable to participate in the transport of the immune complex to the reticuloendothelial system. Instead, tissue deposition of the complex would occur more readily, contributing to the pathologic process. Provided that the early ***complement*** cascade were intact, deficiency of erythrocytic ***CR1*** receptors would contribute to the pathologic response for the same reason.(ABSTRACT ***TRUNCATED*** AT 400 WORDS)

L38 ANSWER 25 OF 37 MEDLINE DUPLICATE 12
AN 90063460 MEDLINE
TI Mapping of the Epstein-Barr virus and C3dg binding sites to a common domain on ***complement*** receptor type 2.
AU Lowell C A; Klickstein L B; Carter R H; Mitchell J A; Fearon D T; Ahearn J M
CS Department of Medicine, Johns Hopkins University School of Medicine, Baltimore, Maryland 21205..
NC AI-22833
AI-28191
5K12-DK01298
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1989 Dec 1) 170 (6) 1931-46.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9003
AB ***Complement*** receptor type 2 (***CR2*** ;CD21), a member of the superfamily of proteins containing short consensus repeats (SCRs), is the B cell receptor for both the gp350/220 envelope protein of Epstein-Barr virus (EBV), and for the C3dg protein of ***complement*** . By analysis of ***CR2*** deletion mutants and ***chimeras*** formed with ***CR1*** (CD35) we determined that of the 15 SCRs in ***CR2*** , the NH2-terminal two SCRs are necessary and sufficient to bind both gp350/220 and C3dg with affinities equivalent to those of the wild-type receptor. The epitope for OKB-7, a mAb that blocks binding of both EBV and C3dg and shares with these ligands B cell-activating capabilities, also requires both SCR-1 and SCR-2, whereas mAbs lacking these functions bind to other SCRs. Thus, EBV, a polyclonal activator of B cells, has selected a site that is proximate or identical to the natural ligand binding site in ***CR2*** , perhaps reflecting the relative immutability of that site as well as its signal transducing function.

L38 ANSWER 26 OF 37 MEDLINE DUPLICATE 13
AN 89036006 MEDLINE
TI Interferon gamma induces synthesis of ***complement*** alternative pathway proteins by human endothelial cells in culture.
AU Ripoche J; Mitchell J A; Erdei A; Madin C; Moffatt B; Mokoena T; Gordon S; Sim R B
CS Department of Biochemistry, Oxford University, United Kingdom..
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1988 Nov 1) 168 (5) 1917-22.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 8902
AB Human umbilical vein endothelial cells grown in vitro under standard conditions contain a high level of mRNA specific for the ***complement*** regulatory factors H and I. An additional 1.8-kb mRNA encoding a ***truncated*** form of factor H is also present. IFN-gamma stimulation of the cells causes a 6-7 fold increase in both factor H mRNA species, and a greater than 10-fold increase in factor I mRNA. IL-1 and LPS slightly suppressed factor H mRNA, while TNF had no effect. mRNA for factor B is also detectable in IFN-gamma-stimulated cells, but messengers for C1q, ***C4bp*** , and CR3 beta chain were not found. Secretion of factor H protein was also stimulated by IFN-gamma. The presence of mRNA for factors H, B, and I, together with C3 secretion, demonstrated by others, suggests that endothelial cells can assemble the complete alternative ***complement*** pathway. Endothelial cell ***complement*** may be involved in leukocyte-endothelium interactions mediated by leukocyte C3 receptors.

L38 ANSWER 27 OF 37 MEDLINE DUPLICATE 14
AN 89010527 MEDLINE
TI Identification of an alternative polyadenylation site in the human C3b/C4b receptor (***complement*** receptor type 1) transcriptional unit and prediction of a secreted form of ***complement*** receptor type 1.
AU Hourcade D; Miesner D R; Atkinson J P; Holers V M
CS Howard Hughes Medical Institute Laboratories, St. Louis, Missouri..
NC 53104
53095
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1988 Oct 1) 168 (4) 1255-70.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
OS GENBANK-Y00812; GENBANK-X14358; GENBANK-X14359; GENBANK-X14360;
GENBANK-X14361; GENBANK-X14362
EM 8901
AB The human C3b/C4b receptor or ***complement*** receptor type one (***CR1***) is an approximately 200-kD single chain membrane glycoprotein of human peripheral blood cells that mediates the binding, processing, and transport of C3b-bearing immune complexes and regulates the activity of the ***complement*** cascade. Analysis of partial cDNA clones has shown that the COOH terminus is composed predominantly of three tandemly repeated regions of 450 amino acids each (15). In this report, we present a cDNA sequence that encodes the NH2 terminus of ***CR1*** . It appears to have been derived from an alternatively processed transcript, caused by

polyadenylation occurring at a site within an intron in the ***CR1*** transcriptional unit. The resulting ***truncated*** messenger carries an open reading frame that would produce a short, secreted ***CR1*** form. We present genomic sequences and Northern blots which support this hypothesis and we propose that the NH2-terminal end of ***CR1*** is a likely location for active sites. In addition, we report evidence for a ***CR1*** -like sequence in the human genome and we present a model for the organization of ***CR1*** .

L38 ANSWER 28 OF 37 MEDLINE DUPLICATE 15
AN 88113786 MEDLINE
TI Structural and functional analysis of ***CR2*** /EBV receptor by means of monoclonal antibodies and limited tryptic digestion.
AU Petzer A L; Schulz T F; Stauder R; Eigentler A; Myones B L; Dierich M P
CS Institute for Hygiene, Innsbruck, Austria..
SO IMMUNOLOGY, (1988 Jan) 63 (1) 47-53.
Journal code: GH7. ISSN: 0019-2805.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 8805
AB The receptor for the C3d fragment of the third component of ***complement*** , ***CR2*** , has recently been shown also to act as the receptor for the Epstein-Barr virus (EBV) and to be involved in the control of B-cell proliferation. In order to define functionally important domains on this molecule, we produced monoclonal antibodies to several distinct epitopes. ***CR2*** was purified from a NP-40 lysate of human tonsils by a new method involving sequential chromatography on lentil lectin Sepharose 4B and DEAE-Sephadex and used to immunize mice. After ***fusion*** we obtained four stable hybridoma lines producing antibody to ***CR2*** . Specificity of these antibodies for ***CR2*** was ascertained by immunofluorescence analysis on a panel of various cells known to possess ***CR2*** , by their reactivity in a recently described ELISA for C3 receptors, by Western blotting with purified ***CR2*** and immunoprecipitation from 125I-labelled Raji cells. These four antibodies were found to recognize three distinct epitopes localized on the same fragments of 95,000, 72,000, 50,000, 32,000 and 28,000 MW obtained after mild tryptic digestion of ***CR2*** . The 72,000 MW fragment contains the binding site for C3d. Two monoclonal antibodies recognizing the same epitope did not inhibit the binding of C3d-coated sheep erythrocytes to Raji cells, whereas the other two antibodies against distinct epitopes did inhibit in the presence of a second antibody. All four monoclonal antibodies stimulated the proliferation of human peripheral blood B cells.

L38 ANSWER 29 OF 37 MEDLINE
AN 87174578 MEDLINE
TI Impaired surface membrane expression of C3bi but not C3b receptors on neonatal neutrophils.
AU Bruce M C; Baley J E; Medvik K A; Berger M
NC HL 31172
SO PEDIATRIC RESEARCH, (1987 Mar) 21 (3) 306-11.
Journal code: OWL. ISSN: 0031-3998.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 8707
AB Because increased ***complement*** receptor expression is necessary for optimal function of adult neutrophils, we tested the hypothesis that the increased susceptibility of neonates to infection might be due to an impaired ability of neonatal neutrophils to increase expression of ***complement*** receptors in response to chemotactic stimuli. We used monoclonal antibodies and flow cytometry to compare surface expression of the receptors for the ***complement*** components C3b (***CR1***) and C3bi (CR3) on adult and neonatal cord blood neutrophils (PMNs). We also compared receptor expression on PMNs from infants delivered by cesarean section without labor versus infants delivered vaginally. Expression of both ***CR1*** and CR3 was minimal on resting adult and neonatal PMNs maintained at 0 degrees C. There was a modest increase in expression of both receptors when PMNs were warmed to 37 degrees C. This increase was similar on adult and neonatal cells, both unfractionated in whole blood and after isolation with Percoll density centrifugation, with one exception. Expression of ***CR1*** was greater on isolated PMNs from vaginally delivered infants versus adults when the cells were warmed to 37 degrees C. This difference was not observed with cells from infants delivered by cesarean section without labor, suggesting this modest increase in receptor expression may be due to factors associated with labor. When isolated cells were stimulated with either N-formyl-methionyl-leucyl-phenylalanine or zymosan-activated serum, expression of ***CR1*** increased to the same extent in both neonatal and adult PMNs. In contrast, maximal CR3 expression on cord PMNs stimulated with N-formyl-methionyl-leucyl-phenylalanine or zymosan-activated serum was only 75% of the adult values.(ABSTRACT ***TRUNCATED*** AT 250 WORDS)

L38 ANSWER 30 OF 37 MEDLINE
AN 87035420 MEDLINE
TI Membrane-bound C4b interacts endogenously with ***complement*** receptor ***CR1*** of human red cells.
AU Kinoshita T; Medof M E; Hong K; Nussenzweig V
NC AI-08499
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1986 Nov 1) 164 (5) 1377-88.
Journal code: I2V. ISSN: 0022-1007.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 8702
AB Activation of the classical ***complement*** pathway on the membrane of autologous cells results in the deposition of C4b on their surface and in the assembly of the C3 convertase C4b2a, one of the amplifying enzymes of the cascade. Here we study the sequence of events leading to irreversible inactivation of the potentially harmful C4b bound to human red cells. We show that deposited C4b interacts endogenously with ***complement*** receptor type 1 (***CR1***) present on the membrane of the same red cell. Complexes containing ***CR1*** and C4b are found in extracts of membranes of C4b-bearing red cells after treatment of the intact cells with a bifunctional crosslinking reagent. The amount of complexed ***CR1*** increases with the number of deposited C4b molecules. Only small amounts of free ***CR1*** are observed on red cells bearing as few as 1,900 molecules of C4b, suggesting that the binding avidity between C4b and endogenous ***CR1*** is high. In agreement with this observation, we find that the deposited C4b inhibits the exogenous cofactor activity of the red cell ***CR1*** for the factor I-mediated cleavage of target-bound clustered C3b. The C4b bound to the human red cells is cleaved by the serum enzyme C3b/C4b inactivator (factor I) and a large fragment (C4c) is released in the incubation medium. The cleavage is totally inhibited by mAbs against ***CR1*** , showing that the ***complement*** receptor is an essential cofactor for the activity of I. When the number of bound C4b per red cell is relatively small (less than 1,000 molecules) the substrate for the enzymatic activity of factor I is mostly or exclusively the C4b bound endogenously to ***CR1*** . Indeed, the kinetics or the extent of cleavage of C4b are not affected by greatly augmenting the concentration of exogenous ***CR1*** or of C4b-bearing red cells in the incubation mixture, thereby increasing the frequency of collisions between ***CR1*** on the surface of one cell with C4b deposited on the membrane of a different cell. On the basis of the present and prior observations, we speculate that both ***DAF*** and ***CR1*** act endogenously to inactivate the function of autologous red cell-bound C4b and prevent the progression of the cascade. ***DAF*** binding prevents the formation of the C3 convertase, C4b2a. The cleavage and irreversible inactivation of C4b only occurs after the concerted activities of endogenous ***CR1*** and serum factor I.(ABSTRACT ***TRUNCATED*** AT 400 WORDS)

L38 ANSWER 31 OF 37 MEDLINE
AN 86203275 MEDLINE
TI Monoclonal anti-human C4b antibodies: stabilization and inhibition of the classical-pathway C3 convertase.
AU Ichihara C; Nakamura T; Nagasawa S; Koyama J
SO MOLECULAR IMMUNOLOGY, (1986 Feb) 23 (2) 151-7.
Journal code: NG1. ISSN: 0161-5890.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 8608
AB Two IgG mouse monoclonal antibodies (MAbs), Abs 242 and 463, were prepared by ***fusion*** of spleen cells from mice immunized with human C4b with a myeloma cell line, P3/ X 63-Ag 8.653. They were assessed for their effect on the activation and stability of the cell-bound classical-pathway C3 convertase, EAC14b2a and on the binding of C2 and ***C4bp*** to EC4b. Ab 242 recognized a conformational neoantigen which appeared upon activation of C4 with C-1s and disappeared after chain separation of C4b, while Ab 463 recognized a linear epitope in the beta-chain of C4b. Ab 242 was found to be a ***C4bp*** -like MAb: it accelerates the decay-dissociation of C3 convertase and interferes with the binding of C2 to C4b. It also interfered with the binding of ***C4bp*** to C4b. These results suggest that Ab 242 recognizes an epitope which is closely related to the C2- and ***C4bp*** -binding sites in C4b. Ab 463, on the other hand, was found to be a nephritic factor like MAb: it prolongs the half-life of C3 convertase from 8 to 30 min at 37 degrees C.

L38 ANSWER 32 OF 37 MEDLINE
AN 87029279 MEDLINE
TI Decreased expression of C3b receptor (***CR1***) on erythrocytes of patients with systemic lupus erythematosus contrasts with its normal expression in other systemic diseases and does not correlate with the occurrence or severity of SLE nephritis.
AU Jouvin M H; Wilson J G; Bourgeois P; Fearon D T; Kazatchkine M D
SO COMPLEMENT, (1986) 3 (2) 88-96.
Journal code: DOB. ISSN: 0253-5076.
CY Switzerland

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 8702
AB Expression of the C3b/C4b receptor (***CR1***) on erythrocytes is decreased in patients with systemic lupus erythematosus (SLE) compared to normal individuals, and the ***CR1*** antigen is absent from podocytes in severe diffuse proliferate nephritis of SLE. In the present study, we examined the relationship between the number of ***CR1*** on erythrocytes and the occurrence and severity of SLE nephritis, and assessed the expression of ***CR1*** on erythrocytes and the occurrence and severity of SLE nephritis, and assessed the expression of ***CR1*** on erythrocytes in non-SLE nephritis and other systemic inflammatory diseases by measuring the binding of 125I-labeled rabbit F(ab')2 and murine monoclonal IgG anti- ***CR1*** antibodies to erythrocytes of normal individuals and patients in a French population. The number of binding sites for monoclonal anti- ***CR1*** antibody on erythrocytes of 116 normal individuals was 743 +/- 22 (mean +/- SEM) with a range of 169-1,333, and the frequency distribution of this number in the population was bimodal. In 112 patients with SLE, the mean number of ***CR1*** sites on erythrocytes was decreased to 62% of the mean for normal individuals (p less than 0.001). No correlation was found between ***CR1*** expression on erythrocytes and the presence or immunohistopathological type of glomerulonephritis in biopsy specimens from these patients. The mean number of ***CR1*** on erythrocytes of 29 patients with non-SLE glomerulonephritis was slightly decreased to 89% of the normal mean (p greater than 0.05), which could not be attributed to glomerular immune complex disease or vasculitis.(ABSTRACT ***TRUNCATED*** AT 250 WORDS)

L38 ANSWER 33 OF 37 MEDLINE
AN 85262899 MEDLINE
TI Disease-associated loss of erythrocyte ***complement*** receptors (***CR1*** , C3b receptors) in patients with systemic lupus erythematosus and other diseases involving autoantibodies and/or ***complement*** activation.
AU Ross G D; Yount W J; Walport M J; Winfield J B; Parker C J; Fuller C R; Taylor R P; Myones B L; Lachmann P J
NC CA 25613
AM 30863
AM 34976
+
SO JOURNAL OF IMMUNOLOGY, (1985 Sep) 135 (3) 2005-14.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 8511
AB Although surface membrane density of ***complement*** receptor type one (***CR1***) on erythrocytes (E) is probably an inherited trait among normal individuals, recent evidence from our laboratories suggests that the reduced number of ***CR1*** per E observed in patients with systemic lupus erythematosus (SLE) results from acquired as well as genetic factors. In the present investigation, the number of ***CR1*** per E was quantitated with 125I-monoclonal anti- ***CR1*** and was found to vary inversely with disease activity in patients with SLE who were followed serially for as long as 14 mo. Although evidence for E surface-bound immune complexes or fixed C3b/iC3b was not obtained, periods of disease activity and low amounts of ***CR1*** per E correlated with the presence of 100 to 800 molecules per E of fixed C3dg fragments (less than 100 C3dg per E in normal subjects). Reduced ***CR1*** and excess fixed C3dg on E also were observed in patients with other disorders associated with ***complement*** activation, including chronic cold agglutinin disease, autoimmune hemolytic anemia, paroxysmal nocturnal hemoglobinuria (PNH), Sjogren's syndrome, and mycoplasma pneumonia. A significant negative correlation (r = -0.498) between ***CR1*** /E and fixed C3dg/E was demonstrable in 255 individual assays evaluated by regression analysis. ***CR1*** decreased and fixed C3dg increased during active disease; the converse was obtained during remission. In patients with active SLE, both serum ***complement*** activity and E ***CR1*** decreased, whereas fixed C3dg fragments increased. By piecewise linear regression analysis, the appearance of 100 to 400 C3dg molecules on patients' E corresponded to a 27 to 60%, reduction in the number of ***CR1*** per E (p less than 0.0002), confirming that fixation of C3 to E was correlated with a loss of ***CR1*** . In patients with PNH, low values for ***CR1*** were observed on moderately ***complement*** -sensitive PNH type II E in association with increased fixed C3 fragments; however, the markedly ***complement*** -sensitive PNH type III E had essentially normal amounts of ***CR1*** and bore little fixed C3. The addition of soluble DNA/anti-DNA immune complexes to normal blood generated levels of fixed C3dg fragments on E comparable to those observed on E from patients with SLE. Kinetic experiments indicated that C3b was fixed to E during the process of immune complex binding and release from E ***CR1*** , and that this fixed C3b was subsequently degraded rapidly to fixed iC3b and more slowly to fixed C3dg without the loss of ***CR1*** that occurs in vivo.(ABSTRACT ***TRUNCATED*** AT 400 WORDS)

L38 ANSWER 34 OF 37 MEDLINE
AN 85209218 MEDLINE
TI Human C5a modulates monocyte Fc and C3 receptor expression.
AU Yancey K B; O'Shea J; Chused T; Brown E; Takahashi T; Frank M M; Lawley T J
SO JOURNAL OF IMMUNOLOGY, (1985 Jul) 135 (1) 465-70.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals; Cancer Journals
EM 8509
AB FcIgG and C3 (***CR1*** and CR3) receptors are responsible for binding opsonized particles, phagocytosis, and immune adherence reactions by circulating and tissue-fixed mononuclear phagocytes. Alterations in the expression of these receptors may thus significantly influence the function of these cells. Because chemoattractants have been shown to both recruit and modulate the function of monocytes, this study specifically examines the effects of human C5a and N-formyl-methionyl-leucyl-phenyl-alanine (FMLP) on human peripheral blood monocyte FcIgG and C3 receptor expression in vitro. Adherent, elutriator-purified monocytes were incubated with C5a (10(-7) to 10(-10) M) or FMLP (10(-5) to 10(-10) M) for 30 min at 37 degrees C, and FcIgG receptor expression was assessed by rosetting with sheep erythrocytes sensitized with limiting dilutions of IgG. Human C5a caused dose-related increases in Fc rosettes of 28% at 10(-9) M, 63% at 10(-8) M, and 167% at 10(-7) M (p less than 0.01). In contrast, no significant increases in monocyte Fc receptor expression were induced by FMLP. Similar rosetting experiments were performed with sheep erythrocytes opsonized with limiting amounts of human C3b to assess C3b receptor expression on adherent human monocytes stimulated with C5a (10(-7) to 10(-10) M) or FMLP (10(-6) to 10(-9) M) for 30 min at 37 degrees C. Again, human C5a caused dose-related increases in monocyte C3b rosette formation; at 10(-8) M and 10(-7) M concentrations of C5a, these increases equaled 119% and 196%, respectively (p less than 0.05). In these experiments, 10(-6) M FMLP also caused a significant increase of 110% in monocyte C3b rosette formation (p less than 0.05). Modulation of monocyte cell surface receptors by human C5a or FMLP was also examined by measuring cell fluorescence and side scatter by dual channel flow cytometry after staining normal leukocytes in citrated venous blood with receptor-specific monoclonal antibodies. These flow cytometric studies demonstrated that both C5a and FMLP induce dose-related increases in ***CR1*** (C3b receptor) and CR3 (iC3b receptor) expression in both monocytes and neutrophils.(ABSTRACT ***TRUNCATED*** AT 400 WORDS)

L38 ANSWER 35 OF 37 MEDLINE
AN 85103718 MEDLINE
TI Deficiency in C3b receptors on neutrophils of patients with chronic granulomatous disease and hyperimmunoglobulin-E recurrent infection (Job's) syndrome.
AU Gaither T A; Gallin J I; Iida K; Nussenzweig V; Frank M M
SO INFLAMMATION, (1984 Dec) 8 (4) 429-44.
Journal code: GM0. ISSN: 0360-3997.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 8505
AB C3b receptor (***CR1***) expression by neutrophils (PMNs) and erythrocytes (Es) from patients with chronic granulomatous disease (CGD) or with hyper-IgE, frequent infection (Job's) syndrome was compared with that of control subjects. The control subjects consisted of one group of patients with infections and a second group of normal, healthy individuals. Three quantitative assays were used: rosette formation with C3b-coated cellular intermediates (EAC43b), binding of radiolabeled monoclonal anti- ***CR1*** ([125I]anti- ***CR1***) to PMN surfaces, and binding of the antibody to nonidet P-40 (NP-40) extracts of PMNs and Es in an immunoradiometric assay. Rosette formation by the PMNs of five male CGD patients was about 50% of that of paired normal control subjects, whereas the rosette formation of three female CGD patients was similar to that of the control subjects. Surface binding of [125I]anti- ***CR1*** to PMNs of 10 CGD patients was about half that of the normal subjects (mean percent binding was 2.33% for the CGD patients vs. 3.86% for the normal subjects, giving a difference of -1.53 +/- 0.22%, P less than 0.001 by the paired-sample t test). The degree of PMN binding was similarly low for both the male and the female CGD patients. Conversely, the binding of anti- ***CR1*** to the PMNs of 11 infected control patients appeared to be similar to that of the normal subjects (4.51% for the patient vs. 4.21% for the paired normal subjects). The infected control group originally included four Job's syndrome patients, and when this subgroup was analyzed separately, their PMNs were shown to bind significantly less anti- ***CR1*** than did the PMNs of the normal subjects (P less than 0.01 by the paired-sample t test). In contrast, the other infected control patients showed higher-than-normal levels of anti- ***CR1*** binding (P less than 0.05). When compared to that of the normal subjects, the total ***CR1*** quantitated in PMN extracts was also lower than normal in CGD patients (P less than 0.01 and in the PMN extracts of eight Job's syndrome patients tested (P less

than 0.01). The PMNs of the other infected control subjects were not significantly different from those of the normal subjects in total ***CR1*** expression. Extracts of Es from Job's syndrome patients also had fewer than normal ***CR1*** (P less than 0.02). On the other hand, ***CR1*** levels in E extracts from the CGD patients and the other control patients were similar to those in the normal control subjects. Quantitations of C3, C4, and factor B were normal in CGD.(ABSTRACT ***TRUNCATED*** AT 400 WORDS)

L38 ANSWER 36 OF 37 MEDLINE
AN 85167122 MEDLINE
TI Protein C.
AU Esmon C T
NC 1 R01 HL30340
5 R01 HL29807
SO PROGRESS IN HEMOSTASIS AND THROMBOSIS, (1984) 7 25-54. Ref: 123
Journal code: Q1B. ISSN: 0362-6350.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 8507
AB The protein C anticoagulant pathway provides many new insights into control mechanisms for regulating coagulation. The observation that protein C deficiency is associated with thrombotic tendencies in the heterozygote (108-109) and early, lethal thrombosis in the homozygote (110, 111) points to the importance of the system as a major regulatory pathway. The complexity of the system has only recently begun to emerge. Thrombin activation of protein C at the endothelial cell surface requires not only the synthesis of thrombomodulin but the coupling of the receptor to a protein C binding site. It is reasonable to assume that an inherited or acquired deficiency in thrombomodulin might lead to thrombotic tendencies. This aspect of the system may explain, in part, the association between vascular disease and thrombosis. Once activated, protein C has an almost total dependence on protein S to express anticoagulant activity. (98) This suggests that deficiencies of protein S may also be associated with thrombotic tendencies. Protein S offers an additional intriguing property. Protein S, a regulatory protein of the coagulation system, is found both free and associated with ***C4BP*** , a regulatory protein of the ***complement*** system. The high affinity, very stable interaction between these components (85) suggests that the interaction is likely to be involved in regulation. (89) The importance of the interaction remains to be demonstrated, but clearly this is a potential direct link between major control proteins of the coagulation and ***complement*** system. Clinical studies suggest that protein C and/or thrombomodulin might be effective therapeutically. Certainly, protein C supplementation during the onset of oral anticoagulant therapy would be expected to circumvent the transient rapid decrease in protein C levels that may influence the early effectiveness of oral anticoagulants. (119) In addition to the systems clinical importance, protein C, its activation, and its function offer a variety of intriguing biochemical problems. For instance, how does thrombomodulin alter the specificity of thrombin? What is the protein C binding site on the cell surface, and what role does Factor Va or its degradation products play in the formation and regulation of this site? How does protein S facilitate activated protein C anticoagulant activity and what roles do membrane surfaces play in this system? What role does beta-hydroxyaspartic acid play in protein C activation and function? How does activated protein C influence fibrinolytic activity? The answers to these questions will undoubtedly add to our understanding of the fundamental mechanisms involved in regulating blood coagulation.(ABSTRACT ***TRUNCATED*** AT 400 WORDS)

L38 ANSWER 37 OF 37 MEDLINE
AN 83110208 MEDLINE
TI Studies of the Epstein Barr virus receptor found on Raji cells. II. A comparison of lymphocyte binding sites for Epstein Barr virus and C3d.
AU Hutt-Fletcher L M; Fowler E; Lambris J D; Feighny R J; Simmons J G; Ross G D
NC CA19014
CA22903
CA25613
+
SO JOURNAL OF IMMUNOLOGY, (1983 Mar) 130 (3) 1309-12.
Journal code: IFB. ISSN: 0022-1767.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 8305
AB A comparison was made between the binding sites of two receptors that are believed to be closely associated on human B lymphocytes: ***complement*** receptor type two (***CR2***) that is specific for C3d fragments, and the receptor (EBVR) for Epstein Barr virus (EBV). Isolated fluid-phase ***CR2*** bound to C3d on erythrocytes (EC3d) and inhibited both B cell-EC3d rosettes and the agglutination of EC3d by anti-C3d, it failed to inhibit either the binding or superinfection of B cells by EBV. By contrast, isolated fluid-phase EBVR inhibited EBV B cell binding activity and superinfection but had no ***CR2*** activity. In addition, radiolabeled ***CR2*** bound to EC3d and anti- ***CR2*** -Sepharose, whereas radiolabeled EBVR did not. Purified fluid-phase C3d fragments inhibited EC3d rosette formation with ***CR2*** +/EBVR+ cells but did not inhibit EBV binding. However, EBV binding to B cells did inhibit EC3d rosette formation. Clones of human/mouse somatic cell hybrids made from ***CR2*** +/EBVR+ human B lymphoblastoid cell and ***CR2*** -/EBVR- mouse myeloma cell parents expressed either EBVR or ***CR2*** but only rarely expressed both EBVR and ***CR2*** . This suggested that the genes for EBVR and ***CR2*** were located on two different human chromosomes. Thus it was concluded that ***CR2*** is probably not the binding site for EBV.

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 *       WELCOME TO THE         *
 *    U.S. PATENT TEXT FILE     *
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=> s (complement receptor 1) or (complement receptor 2) or (decay accelerating
factor) or (membrane cofactor protein) or (c4# binding protein) or (factor H)
      33571 COMPLEMENT
      20995 RECEPTOR
    2108245 1
         11 COMPLEMENT RECEPTOR 1
            (COMPLEMENT(W)RECEPTOR(W)1)
      33571 COMPLEMENT
      20995 RECEPTOR
    2105705 2
          5 COMPLEMENT RECEPTOR 2
            (COMPLEMENT(W)RECEPTOR(W)2)
      26425 DECAY
      36012 ACCELERATING
     220458 FACTOR
         41 DECAY ACCELERATING FACTOR
            (DECAY(W)ACCELERATING(W)FACTOR)
      72436 MEMBRANE
       1403 COFACTOR
      50144 PROTEIN
          6 MEMBRANE COFACTOR PROTEIN
            (MEMBRANE(W)COFACTOR(W)PROTEIN)
      20530 C4#
      86467 BINDING
      50144 PROTEIN
         45 C4# BINDING PROTEIN
            (C4#(W)BINDING(W)PROTEIN)
     220458 FACTOR
     536622 H
        698 FACTOR H
            (FACTOR(W)H)
L1       752 (COMPLEMENT RECEPTOR 1) OR (COMPLEMENT RECEPTOR
2) OR (DECA
Y A
            CCELERATING FACTOR) OR (MEMBRANE COFACTOR PROTEIN)
OR (C4#
BIN
            DING PROTEIN) OR (FACTOR H)

=> s l1 and protein
      50144 PROTEIN
L2       106 L1 AND PROTEIN

=> s hybrid? or chimer? or fusion or truncat?
      38190 HYBRID?
       1799 CHIMER?
      34948 FUSION
      36258 TRUNCAT?
L3     101996 HYBRID? OR CHIMER? OR FUSION OR TRUNCAT?

=> s l2 (p) l3
*WARNING* - PROXIMITY OPERATOR PRECEDENCE LEVEL CONFLICTS
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FIELD CODE - 'AND' OPERATOR ASSUMED 'L2 (P) L3'
L4       82 L2 (P) L3

=> s l3 (2a) protein
      50144 PROTEIN
L5     2435 L3 (2A) PROTEIN

=> s l1 (p) l5
L6        5 L1 (P) L5

=> d bib date ab 1-


US PAT NO:  5,525,708 [IMAGE AVAILABLE]          L6: 1 of 5
```

DATE ISSUED: Jun. 11, 1996
TITLE: Covalent dimer of kit ligand
INVENTOR: Karl H. Nocka, Harvard, MA
Robert B. Lobell, Watertown, MA
ASSIGNEE: CytoMed, Inc., Cambridge, MA (U.S. corp.)
APPL-NO: 08/220,379
DATE FILED: Mar. 28, 1994
ART-UNIT: 182
PRIM-EXMR: Marianne P. Allen
LEGAL-REP: James F. Haley, Jr., Andrew S. Marks

L6: 1 of 5

TITLE: Covalent dimer of kit ligand
US PAT NO: 5,525,708 DATE ISSUED: Jun. 11, 1996
[IMAGE AVAILABLE]
APPL-NO: 08/220,379 DATE FILED: Mar. 28, 1994

ABSTRACT:
A modified form of KL, the ligand for the c-Kit proto-oncogene, has been prepared wherein the protein is stabilized by an intermolecular covalent linkage. The protein can be prepared by expression of a recombinant protein which is dissolved in denaturant and refolded under conditions resulting in a disulfide linked dimer. Examples demonstrate the purification and characterization of this disulfide-linked cysteine dimer kit ligand (KL-CD) which contains at least one intermolecular disulfide bond and has at least ten-fold greater activity in promoting cell proliferation than native, non-covalently linked KL, as measured in in vitro assays.

US PAT NO: 5,328,891 [IMAGE AVAILABLE] L6: 2 of 5
DATE ISSUED: Jul. 12, 1994
TITLE: Insulin-like growth factor binding protein and pharmaceutical compositions
INVENTOR: Robert C. Baxter, Glebe, Australia
William I. Wood, San Mateo, CA
ASSIGNEE: Genentech, Inc., S. San Francisco, CA (U.S. corp.)
Central Sydney Area Health Service, Camperdown, Australia (foreign corp.)
APPL-NO: 08/003,710
DATE FILED: Jan. 13, 1993
ART-UNIT: 182
PRIM-EXMR: Robert J. Hill, Jr.
ASST-EXMR: Marianne Porta Allen
LEGAL-REP: Janet E. Hasak

L6: 2 of 5

TITLE: Insulin-like growth factor binding protein and pharmaceutical compositions
US PAT NO: 5,328,891 DATE ISSUED: Jul. 12, 1994
[IMAGE AVAILABLE]
APPL-NO: 08/003,710 DATE FILED: Jan. 13, 1993
REL-US-DATA: Division of Ser. No. 171,623, Mar. 22, 1988, Pat. No. 5,258,287.

ABSTRACT:
DNA isolates coding for insulin-like growth factor binding protein may be used to produce the protein via recombinant expression systems. Insulin-like growth factor binding protein, which generally has a molecular weight of about 53 kD on non-reducing SDS-PAGE, is useful as a binder to insulin-like growth factor and as a metabolic regulator.

US PAT NO: 5,258,287 [IMAGE AVAILABLE] L6: 3 of 5
DATE ISSUED: Nov. 2, 1993
TITLE: DNA encoding and methods of production of insulin-like growth factor binding protein BP53
INVENTOR: Robert C. Baxter, Plebe, Australia
William I. Wood, San Mateo, CA
ASSIGNEE: Genentech, Inc., South San Francisco, CA (U.S. corp.)
Central Sydney Area Health Service, Camperdown, Australia (foreign corp.)
APPL-NO: 07/171,623
DATE FILED: Mar. 22, 1988
ART-UNIT: 182
PRIM-EXMR: Robert J. Hill, Jr.
ASST-EXMR: Marianne Porta Allen
LEGAL-REP: Janet E. Hasak

L6: 3 of 5

TITLE: DNA encoding and methods of production of insulin-like growth factor binding protein BP53
US PAT NO: 5,258,287 DATE ISSUED: Nov. 2, 1993
[IMAGE AVAILABLE]
APPL-NO: 07/171,623 DATE FILED: Mar. 22, 1988

ABSTRACT:
DNA isolates coding for insulin-like growth factor binding protein may be used to produce the protein via recombinant expression systems. Insulin-like growth factor binding protein, which generally has a molecular weight of about 53 kD on non-reducing SDS-PAGE, is useful as a binder to insulin-like growth factor and as a metabolic regulator.

US PAT NO: 5,242,687 [IMAGE AVAILABLE] L6: 4 of 5
DATE ISSUED: Sep. 7, 1993
TITLE: Method of reducing cellular immune response involving T-cells using CD8-bearing antigen presenting cells
INVENTOR: Mark L. Tykocinski, Shaker Heights, OH
David R. Kaplan, Cleveland Heights, OH
ASSIGNEE: TKB Associates Limited Partnership, Cleveland, OH (U.S. corp.)
APPL-NO: 07/691,475
DATE FILED: Apr. 25, 1991
ART-UNIT: 183
PRIM-EXMR: Christine M. Nucker
ASST-EXMR: T. Cunningham
LEGAL-REP: Lyon & Lyon

L6: 4 of 5

TITLE: Method of reducing cellular immune response involving T-cells using CD8-bearing antigen presenting cells
US PAT NO: 5,242,687 DATE ISSUED: Sep. 7, 1993
[IMAGE AVAILABLE]
APPL-NO: 07/691,475 DATE FILED: Apr. 25, 1991
REL-US-DATA: Continuation of Ser. No. 429,401, Oct. 31, 1989, abandoned, and a continuation-in-part of Ser. No. 323,770, Mar. 15, 1989, abandoned.

ABSTRACT:
Specific and nonspecific immunomodulation, enhancement of cellular engraftment, and modulation of nonimmune cells are achieved by using various membrane-binding and soluble CD8 compositions.

US PAT NO: 5,223,394 [IMAGE AVAILABLE] L6: 5 of 5
DATE ISSUED: Jun. 29, 1993
TITLE: Recombinant DNA molecule comprising lymphocyte function-associated antigen 3 phosphatidylinositol linkage signal sequence
INVENTOR: Barbara P. Wallner, Cambridge, MA
ASSIGNEE: Biogen, Inc., Cambridge, MA (U.S. corp.)
APPL-NO: 07/335,688
DATE FILED: Apr. 10, 1989
ART-UNIT: 182
PRIM-EXMR: Robert J. Hill, Jr.
ASST-EXMR: Laurie Scheiner
LEGAL-REP: James F. Haley, Jr., Denise L. Loring, Immac J. Thampoe

L6: 5 of 5

TITLE: Recombinant DNA molecule comprising lymphocyte function-associated antigen 3 phosphatidylinositol linkage signal sequence
US PAT NO: 5,223,394 DATE ISSUED: Jun. 29, 1993
[IMAGE AVAILABLE]
APPL-NO: 07/335,688 DATE FILED: Apr. 10, 1989

ABSTRACT:
DNA sequences derived from a phosphatidylinositol-linked form of lymphocyte function-associated antigen 3 ("LFA-3") are provided which code for a phosphatidylinositol linkage signalling sequence. The linkage signalling sequence may be linked to DNA coding for secretory proteins or polypeptides to obtain phosphatidylinositol-linked chimeric proteins or polypeptides. The chimeric proteins can be used to produce targeted drugs, to form micellular or liposomal drug delivery systems, or to improve the purification or screening of particular cells, proteins or DNA libraries.

=> d kwic 1-

US PAT NO: 5,525,708 [IMAGE AVAILABLE] L6: 1 of 5

DETDESC:

DETD(22)

Methods for making soluble dimeric protein which is expressed on the host cell surface as a **chimeric** **fusion** **protein** incorporating the extracellular portion of the protein with the stem region of **C4b** **binding** **protein** (C4bp) are described in U.S. Ser. No. 08/118,366 filed Aug. 8, 1993,

DETDESC:

DETD(23)

In these constructs, the extracellular domain of CD28, a cell surface dimer, is fused to C-terminal 58 amino acids of **C4** **binding** **protein**. When these constructs are expressed in mammalian cells, a multimeric CD28-C4bp protein is expressed on the cell surface. The protein. . . amino acids of PAP. When this construct is expressed, the C-terminal 30 amino acids of the PAP portion of the **fusion** **protein** are cleaved, leaving a C-terminal arginine residue. That arginine is then available for the addition of phosphatidylinositol glycan (pI-G). The pI-G acts as an anchor to hold the **fusion** **protein** to the cell membrane. Subsequent cleavage with phospholipase

C releases soluble, dimeric CD28.

US PAT NO: 5,328,891 [IMAGE AVAILABLE] L6: 2 of 5

DETDESC:

DETD(9)

Fusions . . . from the normal cell surface receptor for IGF-I or IGF-II, or the phospholipid anchor domain at the C-terminus of mature **decay** **accelerating** **factor** (mDAF) described in EPO Pub. No. 244,267 published Nov. 4, 1987, the disclosure of which is incorporated herein by reference,. . . BP-53 may be expressed in recombinant cell culture as a C-terminal fusion of the pre-BP53 with mDAF. For example, a **fusion** **protein** may be constructed in which the last 37 amino acids of membrane DAF predicted by the spliced cDNA is fused. . .

US PAT NO: 5,258,287 [IMAGE AVAILABLE] L6: 3 of 5

DETDESC:

DETD(9)

Fusions . . . from the normal cell surface receptor for IGF-I or IGF-II, or the phospholipid anchor domain at the C-terminus of mature **decay** **accelerating** **factor** (mDAF) described in EPO Pub. No. 244,267 published Nov. 4, 1987, the disclosure of which is incorporated herein by reference,. . . BP-53 may be expressed in recombinant cell culture as a C-terminal fusion of the pre-BP53 with mDAF. For example, a **fusion** **protein** may be constructed in which the last 37 amino acids of membrane DAF predicted by the spliced cDNA is fused. . .

US PAT NO: 5,242,687 [IMAGE AVAILABLE] L6: 4 of 5

DETDESC:

DETD(20)

Linear . . . linked in-frame to the coding sequence for the 3' end of a protein that naturally undergoes glycoinositolphospholipid modification, such as **decay** **accelerating** **factor** (DAF). The **chimeric** **protein** produced in this way undergoes glycoinositolphospholipid modification inside the cell. This glycoinositolphospholipid-modification process was discovered by one of the inventors. . . for the chimeric gene transcriptional cassette, vectors and host cells will dictate the nature of post-translational modifications introduced into the **chimeric** **protein** and the quantity of protein produced. For instance, baculovirus promoters and vectors can be used in insect host cells to. . .

US PAT NO: 5,223,394 [IMAGE AVAILABLE] L6: 5 of 5

SUMMARY:

BSUM(7)

The . . . a signal for phosphatidylinositol attachment. In one study, DNA coding for the 37-amino acid C-terminal sequence of the PI-linked protein, **decay** **accelerating** **factor** (DAF), was fused to the 3' end of DNA coding for a normally secreted protein fragment of glycoprotein D (from herpes simplex virus-1), resulting in a PI-linked **fusion** **protein**. See, Caras et al., Science 238, 1280-1283 (1987). However, comparison of the C-terminal sequences of many precursors of PI-linked proteins. . .

=> log y

U.S. Patent & Trademark Office LOGOFF AT 16:10:06 ON 09 JAN 97